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FIG. 1.

Lupus vulgaris.

FIG. 2.

Section of breast, showing carcinoma.

(*Page 175.*)

INTERNATIONAL CLINICS

A QUARTERLY

OF

ILLUSTRATED CLINICAL LECTURES AND
ESPECIALLY PREPARED ORIGINAL ARTICLES

ON

TREATMENT, MEDICINE, SURGERY, NEUROLOGY, PÆDIATRICS, OBSTETRICS, GYNÆCOLOGY, ORTHOPÆDICS, PATHOLOGY, DERMATOLOGY, OPHTHALMOLOGY, OTOLOGY, RHINOLOGY, LARYNGOLOGY, HYGIENE, AND OTHER TOPICS OF INTEREST TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION
THROUGHOUT THE WORLD

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CONTENTS OF VOLUME IV

(NINETEENTH SERIES)

TREATMENT

PAGE

ANTIMENINGITIS SERUM AND THE RESULTS OF ITS EMPLOYMENT. By Simon Flexner, M.D. 1

TREATMENT OF CANCER BY FULGURATION. By Pierre Fredet, M.D. 22

THE USE OF TUBERCULIN IN TREATMENT. By Louis Hamman, M.D. 31

GENERAL CONSIDERATION OF THE THERAPEUTIC USES OF DIET. By George M. Niles, M.D. 54

HYPNOTISM AND PSYCHOTHERAPY. By J. W. Wainwright, M.D... 60

MEDICINE

DIAGNOSIS AND TREATMENT OF PERNICIOUS ANÆMIA. By Walter L. Bierring, M.D. 67

ADRENAL INSUFFICIENCY. By Emile Sergent, M.D. 88

PROGNOSIS IN ADVANCED TUBERCULOSIS. By William B. Stanton, M.D. 99

GOUTY PHLEBITIS. By Sir Dyce Duckworth, BT., M.D., LL.D., F.R.C.P 108

THE DUTY OF THE PHYSICIAN IN PREVENTION OF ORGANIC DISEASE OF THE HEART. By Henry M. Goodman, A.B., M.D..... 114

SURGERY

REPORT OF FIFTEEN CASES OPERATED UPON IN PROFESSOR W. L. RODMAN'S SURGICAL CLINIC AT THE MEDICO-CHIRURGICAL HOSPITAL. By J. Stewart Rodman, M.D. 125

EPIPLOITIS FOLLOWING THE RADICAL CURE OF HERNIA. By Charles Green Cumston, M.D. 145

COLOR PHOTOGRAPHS IN RELATION TO SURGERY. By C. B. Longenecker, M.D. 166

THE USE OF ETHYL CHLORIDE AS A GENERAL ANÆSTHETIC. By W. Estell Lee, M.D. 182

RÖNTGENOLOGY

THE RÖNTGEN DIAGNOSIS OF PULMONARY TUBERCULOSIS. By Charles Lester Leonard, M.D., A.M. 189

GYNÆCOLOGY AND OBSTETRICS

PAGE

THE EARLY DIAGNOSIS OF CANCER OF THE UTERUS; OPERATIVE TECHNIC. By Thomas S. Cullen, M.B. 193

GRAVITY METHOD OF REMOVING ADHERENT PLACENTA. By J. J. Rectenwald, M.D. 199

OVARIAN FIBROMATA. By C. C. Mapes, M.D. 205

GENITO-URINARY DISEASES

THE INDICATIONS FOR SURGERY OF THE PROSTATE. By John B. Deaver, M.D., LL.D. 228

PÆDIATRICS

OBSERVATION ON INFANTILE TYPHOID FROM THE BABIES' WARD OF THE SYDENHAM HOSPITAL. By Louis Fischer, M.D.. 242

NOTES ON THE DIAGNOSIS AND TREATMENT OF CONGENITAL SYPHILIS. By G. R. Pisek 247

PARASITOLOGY

HYMENOLEPIS NANA IN THE UNITED STATES, WITH REPORT OF TWO CASES. By William H. Deaderick, M.D. 252

FRAMBŒSIA TROPICA. By Paul G. Woolley, M.D. 262

LARYNGOLOGY

DIAGNOSIS OF FOREIGN BODIES OF THE ŒSOPHAGUS AND BRONCHI. By G. Guisez, M.D. 272

PATHOLOGY

DEATH DUE TO LEUKÆMIC INFILTRATION OF THE LARYNX; PRIAPISM PERSISTENT POST MORTEM, MYELOID THROMBOSES IN THE CORPORA CAVERNOSA; FATAL HEMORRHAGE FROM SPLEEN AFTER PROLONGED RADIOTHERAPY. By Aldred Scott Warthin, Ph.D., M.D. 280

A SOLID TERATOMA OF THE MEDIASTINUM WITH HISTORY AND AUTOPSY NOTES. By E. W. Becker, M.D., and H. W. Carey, A.B., M.D. 296

LIST OF ILLUSTRATIONS TO VOLUME IV

(NINETEENTH SERIES)

COLORED PLATES

PAGE

Lupus vulgaris (Figure 1), section of breast showing carcinoma (Figure 2) ..... *Frontispiece*
Photo-micrograph of carcinoma of the breast (Fig.3) 176
Gangrene of foot (Fig. 4) 176

PLATES

Section of spinal cord from case of aplastic pernicious anæmia (Fig. 1)... 74
Section of medulla from case of aplastic pernicious anæmia (Fig. 2)..... 74
Omental cyst (Figs. 1 and 2)..... 126
Congenital angioma of face (Fig. 3)..... 130
Unilateral cystic goitre (Figs. 4 and 5) 131
Semilateral cystic goitre (Fig. 6) 132
Unilateral cystic goitre (Fig. 7) 132
Operative result in adenofibroma of breast (Fig. 8)..... 134
Carcinoma of the breast (Fig. 9)..... 135
Operative result in carcinoma of the breast (Fig. 10)..... 135
Anastomosis of ulnar and median nerves in the wrist (Figs. 11 and 12) ... 142
Lupus vulgaris (Fig. 5)..... 178
Carcinoma of the breast (Fig. 6) 178
Section of breast showing carcinoma (Fig. 7) 178
Photo-micrograph of carcinomatous axillary node (Fig. 8) 178
Postoperative involvement of chest by carcinoma (Fig. 9) 178
Skiagraphs showing tuberculous bronchial adenitis (Figs. 1 and 2) 190
Skiagraph showing tuberculous infiltration of right apex of lung (Fig. 3).. 190
Skiagraph showing tuberculous peribronchial infiltration (Fig. 4) 190
Skiagraph showing tuberculous consolidation of lung (Fig. 5) 190
Skiagraph showing consolidated left lung with cavity in apex (Fig. 6).... 191
Skiagraph showing tuberculous consolidation and cavity in left upper lobe (Fig. 7) 192
Skiagraph showing tuberculous infiltration and softening of lungs (Fig. 8). 192
Skiagraph showing peribronchial scar-tissue (Fig. 9) 192
Skiagraph showing localized non-tuberculous empyema (Fig. 10) 192
Skiagraph showing symmetrical pulmonary congestion (Fig. 11) 192
Myeloid thromboses in corpora cavernosa (Figs. 1 and 2) 288

FIGURES

PAGE

Apparatus for treatment of cancer by fulguration (Fig. 1) 23
Chart of blood findings in pernicious anæmia (Fig. 3) 80
Chart of blood findings in pernicious anæmia (Fig. 4) 81
Chart of blood findings in pernicious anæmia (Fig. 5) 84
Chart of blood findings in pernicious anæmia (Fig. 6) 85
Temperature chart in typhoid fever (Fig. 1) 244
Temperature chart in typhoid fever (Fig. 2) 245
Hymenolepis nana (Fig. 1) 255
Scolex of Hymenolepis nana (Fig. 2) 255
Scolex of Hymenolepis nana (Fig. 3) 256
Scolex of Hymenolepis nana (Fig. 4) 257
Hooklets of Hymenolepis nana (Fig. 5) 258
Proglottides of Hymenolepis nana (Fig. 6) 258
Proglottides of Hymenolepis nana containing ova (Fig. 7) 259
Ova of Hymenolepis nana (Figs. 8 and 9) 260

Treatment

ANTIMENINGITIS SERUM AND THE RESULTS OF ITS EMPLOYMENT

BY SIMON FLEXNER, M.D.

FROM THE LABORATORIES OF THE ROCKEFELLER INSTITUTE FOR MEDICAL RESEARCH
NEW YORK

The epidemic of meningitis which prevailed in America and Europe from 1904 to 1908 will be regarded as especially significant because during its prevalence the final proof was brought that Diplococcus intracellularis of Weichselbaum was the sole cause of epidemic meningitis in man, and the discovery of the efficacy of antimeningococcic serum in the treatment of that malady was made. It cannot be regarded as remarkable, in view of the appalling mortality and the severe consequences of the disease, and of the absence of all really efficient means of combating it, that an effort should be made to affect favorably its course by means of an antiserum. It could, of course, be predicted that the immunization of animals with cultures of Diplococcus intracellularis would lead to the development of an antiserum of some degree of potency, but whether this antiserum would prove of any value in the treatment of epidemic meningitis could not, obviously, be foreseen.

In the pursuit of this idea two methods of proceeding were available: the antiserum could be produced and then applied empirically to the treatment of the disease, or an experimental basis might first be sought upon which the employment of the antiserum in man might ultimately come to be based. The first, or empirical method, was, as a matter of fact, followed to a large extent in Germany by Jochmann, and by Kolle and Wassermann, while the second, or experimental method, was employed in the United States by Flexner. The difference in the early results obtained in the serum treatment of epidemic meningitis in the two

countries is striking and significant and can be attributed only to the difference in the manner of its development. It is desirable to recall here for a moment that the injection of living cultures of Diplococcus intracellularis into the serous cavities of small animals such as mice, guinea-pigs and rabbits, leads to an intoxication and rarely to an infection causing their death. The experimental effects thus produced are highly dissimilar to the lesions present in the natural disease in man. As a consequence, the mere combating of this experimental disease by means of an antiserum would not afford an adequate support for the employment of the antiserum in man. It is, however, possible to produce in certain species of lower monkeys, among which Macacus rhesus was shown to be well adapted, an acute leptomeningitis by the direct injection of active cultures into the subdural space of the spinal cord which presents the lesions, and sometimes the symptoms corresponding to those present in the natural disease in man. This being true, it is a fair assumption that if the antiserum is capable of controlling such an infection, its favorable action in the natural disease in man may be fairly predicted. This important basis for the employment of the antimeningitis serum in the natural infection in man resulted from the experiments of Flexner.

PREPARATION AND STANDARDIZATION OF THE ANTIMENINGITIS SERUM

Horses are employed for preparing the antimeningitis serum for the treatment of epidemic meningitis in man. Cultures of Diplococcus intracellularis and an autolysate prepared from the cultures are injected alternately. The diplococcus is grown on large flat surfaces in flasks of about one-half litre capacity on a medium composed of beef infusion agar-agar containing 2 per cent. of glucose. The cultures, of which many different strains are used, when employed for injection or for preparing the autolysate, should have been incubated at 37° C. for twenty-four hours. The cultures for injection are suspended in normal, sterile, saline solution. The autolysate is prepared by pouring over the surface of the growth 10 to 20 c.c. of sterile, normal salt solution to which is afterwards added about 1 per cent. of toluol. By gently agitating the flask from side to side the toluol and salt solution are mixed

and the cultures separated in part from the medium. The flask is returned to the thermostat where it is allowed to remain for another twenty-four hours, during which time the toluol has evaporated. The resulting fluid remaining contains no diplococci but some granular débris and a solution of the micro-organisms. This fluid autolysate is subjected to centrifugalization or is employed for injection without further treatment. Placed in sterile flasks, it can be kept for purposes of immunization for several weeks in the refrigerator. The destruction of the diplococci is effected through the agency of an active autolytic ferment.

The practical operation of immunization of horses is carried out by the employment of cultures of the meningococcus killed by heat at 60° C. or in the living state, which are injected alternately with the autolysate. The injections are made once a week into the subcutaneous tissues. Owing to the severe action of the cultures and the autolysate upon the organs of circulation, the employment of intravenous injections is very dangerous and does not seem to give a more potent antiserum. The subcutaneous injections should be made at several separate places at each operation so as to extend the area of antibody formation in the tissues and to reduce the intensity of the local reaction. The doses should at first not be too large since it is desirable to prevent very severe local reactions and high febrile rise in temperature. The usual temperature observed on the day following the inoculations varies from 39° to 39.6° C. and is maintained for about one day. The local swelling, which may extend from the shoulder to the knee, sets in in a few hours, reaches its height in forty-eight hours or less, and disappears usually in a few days. The larger inoculations, especially of the living cultures, may lead to the production of sterile abscesses. Beginning with a fraction of a flask of the culture and a corresponding amount of the autolysate, the quantities may be gradually increased until ultimately the contents of many flasks may be given at a single inoculation. During the first month or two of inoculation it is advisable to use killed cultures and after that living cultures to alternate with the autolysate. When the period of bleeding has arrived the inoculations are made at the proper intervals to alternate with the bleeding. It is advisable to continue the process of immunization from four to six months before beginning to collect serum for

therapeutic use. The precise period at which the collection of serum can be begun will depend upon the rapidity with which the immunity principles are developed. This is determined by the standardization of the serum.

The antimeningitis serum, being of the general nature of an anti-endotoxic serum, is not subject to standardization according to the methods employed with the antitoxic sera. The antimeningitis serum possesses, it is true, a certain but definite neutralizing power for the autolysate, but this property cannot be used for purposes of titration. The anti-endotoxic strength of the serum is probably not a correct measure of its therapeutic value since it is not a measure of other immunity principles contained within the serum, whose action is very important. Moreover, the autolysate does not act in a uniform manner upon young guinea-pigs and mice, which are, on the whole, the laboratory animals most susceptible to its toxic effects. Whether introduced beneath the skin, into the peritoneal cavity, or, as tested by Jobling, by intracardiac injection, the fatal dose fluctuates greatly in guinea-pigs of 200 grammes weight. The employment of living cultures of the meningococcus for purposes of standardization of the serum is also excluded, by reason of their highly variable pathogenicity, for mice, guinea-pigs and other animals. Nor do we possess at present any means by which the pathogenic properties of the living cultures can be maintained at a fixed and sufficiently high level. Moreover, no fixed relation exists between the complement-binding power of the antiserum and its therapeutic activity, and hence this method of titration, introduced by Kolle and Wassermann, has had to be abandoned. On the other hand, it now seems not improbable that in the opsonins or bacteriotropins we possesses a measure of the therapeutic activity of the antimeningitis serum. It is not to be supposed that the activity of the antiserum resides wholly in the opsonins, but it is probable that they are a measure of other useful immunity principles present, just as they undoubtedly come directly into operation in the course of the therapeutic action of the antiserum. Neufeld first proposed to use the opsonins as an index of activity of the antiserum, and a convenient method of standardization based upon the opsonins has been given by Jobling.

The leucocytes are obtained from the peritoneal cavity of a

guinea-pig by injecting an aleuronat suspension one day and collecting the exudate the next day. They are then washed four times in normal salt solution after which they are suspended in saline solution in such amount as to equal in opacity a 0.3 per cent. lecithin emulsion. The suspension of the meningococcus is prepared by adding one c.c. of an equal mixture of bouillon and salt solution to a slanting culture-tube of a twenty-four-hour growth of the diplococcus. A suitable culture of the meningococcus, which does not disintegrate too readily in the leucocytes and is readily taken up by them, must be chosen. The serum dilutions and diplococci are to be incubated at 37° C. for one hour before the leucocytes are added. After they have been added, the whole is again incubated for thirty minutes, after which the films are to be prepared. The latter are then stained with a suitable aniline dye and subjected to microscopical examination. The readings are based on the gross appearance of the films, as recommended by Neufeld. The greatest dilution at which the antiserum produces a degree of phagocytosis exceeding the control preparations in the saline solution alone represents the value of the antiserum. The greatest dilution of the antiserum tested by Jobling, at which a definite increase in phagocytosis has been noted, is about 1 to 20,000. Normal horse serum usually has its phagocytic powers exhausted at a dilution of about 1 to 100. A study of several different therapeutically active antimeningococcic sera has led Jobling to the conclusion that a suitable standard of opsonic strength for such an antiserum is that in which a dilution of five thousand times still shows a degree of phagocytic activity exceeding the saline controls. Hence a standard serum should have the value of five thousand expressed in opsonic units. The opsonins or bacteriotropins of the antiserum appear to be highly durable and to persist unchanged, under proper conditions of refrigeration, for one year or longer.

MANNER OF THERAPEUTIC ACTION

The antimeningitis serum possesses direct and indirect bactericidal power. In common with normal serum and sterile exudates, but in a greater degree than these, it exercises direct bactericidal effect on cultures of the meningococcus. This action is entirely independent of the presence of complement, but is influenced greatly

by the degree of concentration of the serum. Weak dilutions have little or no effect; strong concentrations have a marked effect (Flexner).

The antiserum promotes phagocytosis of the meningococcus *in vitro,* as was first shown by Jochmann and others, and in the animal body, as was noted by Flexner and Kolle and Wassermann. Moreover, the meningococci which have been englobed by leucocytes, after being acted upon by the immune serum, are more quickly and readily disintegrated than when not thus acted upon (Flexner). The antiserum also possesses a certain, although moderate, antitoxic power through which it is rendered capable of neutralizing larger quantities of the toxic autolysate than is normal serum. The meningococci when destroyed by intracellular digestion appear at the same time to be detoxicated (Flexner), hence the antimenigitis serum may be regarded as producing its beneficial therapeutic effects through: (*a*) its power to act directly upon, and to inhibit the growth of, or to destroy outright the meningococcus; (*b*) its property of increasing phagocytosis and promoting intracellular digestion of the diplococcus, and (*c*) its power of neutralizing the soluble toxic products set free by disintegration of the meningococcus. Experiments carried out in the animal body and supported by those made *in vitro* show that these several forms of therapeutic activity of the antiserum could be secured best provided the antiserum was brought directly, and in concentrated form, into relation with the focus of meningococcus infection and inflammation. The favorable action of the antiserum under these conditions was especially noticeable in the experimental form of cerebrospinal meningitis in monkeys, produced by subdural inoculations of cultures of the meningococcus. Hence the successful employment of the antiserum in human beings is dependent upon its injection into the subdural space.

The objective effects of the intralumbral injection of the antiserum in human beings suffering from epidemic meningitis are readily demonstrable. Very soon after the injection, the meningococci tend to be reduced in number in the exudate, to disappear from its fluid parts, to be taken up partly or wholly by leucocytes, and to become swollen or fragmented, and to stain diffusely and indistinctly and at the same time to lose the capacity for growing in

cultures. It is a common experience to find that even when some of the diplococci still remain extracellular, while many more are contained within leucocytes, that their cultivation outside the body has been rendered impossible by one or two injections of the antiserum. Moreover, the leucocytes containing the altered meningococci rarely show the evidences of degeneration such as are seen not infrequently when the diplococci still stain sharply and remain viable in cultures, as happens when the antiserum has not been employed.

Along with the destructive influences of the antiserum on the diplococci go certain favorable changes in the inflammatory exudate, through which it loses turbidity irrespective of whether it is only cloudy or whether it is already purulent. The rapidity with which the purulent exudate may clear up under the influence of the serum injections is very striking. Closely associated with the loss of leucocytes by the cerebrospinal exudate and its return to a limpid condition is the state of the general leucocytes of the blood, which in favorable cases going on to recovery fall rapidly and even critically in number. When, as sometimes and not very frequently happens, the meningococci are not restrained in multiplication, and are not altered so as to undergo rapid phagocytosis and intracellular digestion but tend to be resistant, the exudate may not clear up but may become even more purulent and the diplococci may multiply, while the leucocytes in the general circulation instead of falling actually increase in number.

INDICATIONS FOR THE EMPLOYMENT OF THE ANTISERUM

The employment of the antiserum is indicated in all forms of meningococcic infection. Since Diplococcus intracellularis is concerned chiefly, as far as our present knowledge warrants a conclusion, with the production of epidemic cerebrospinal meningitis, its use is indicated chiefly in that disease. However, other localized local meningococcic infections may also be subject to treatment by the antiserum. This would appear especially true of the metastatic joint infections not infrequently attending epidemic meningitis. After aspiration of the inflammatory exudate, the direct injection of the antiserum into the inflamed joint has been followed, as reported by Ladd, by rapid improvement in the condition. It is even possible that the rare instances of meningococcic endocarditis,

described by Warfield and by Weichselbaum, which can be diagnosed during life by means of blood cultures, may be benefited by intravenous injections of the antiserum. Already two cases have been reported, one by Bovaird and the other by Netter, in which a general blood infection with the meningococcus existed without discoverable local inflammation of the cerebrospinal meninges or other organs, attended by great mental excitement, and in which the intravenous injection of the serum was followed by recovery. These observations encourage the view that the endocardial infections may also be favorably influenced, but they are particularly interesting in view of the question still unsettled as to the portal of entry of the meningococcus into the body and the manner of infection of the cerebrospinal meninges. Although there are many grounds for supposing that the meninges are infected directly by the diplococci passing by way of the lymphatics of the nasal mucosa to the lymphatics of the meninges, yet the possibilities of blood infection have not been entirely excluded. However, it is especially in the meningococcal infections of the meninges that the use of the antiserum is indicated, and in this disease every effort should be made to inject the serum early. It is even advisable to employ the antiserum in some cases of meningitis of doubtful etiology pending the making of a bacteriological diagnosis under circumstances which are suspicious.

MANNER OF ADMINISTERING THE ANTISERUM

Since the efficacy of the serum depends in considerable part upon the degree of its concentration at the seat of the disease, it should be employed only by subdural injection. The serum passes very poorly from the blood into the spinal fluid, and even if it passed readily the high degree of dilution which it suffers in the blood would prevent its accumulation in the meninges in a high state of concentration. The antiserum is readily injected into the subdural space and the risk of producing a dangerous increase of intradural pressure from the injection is so small as to be negligible, especially as in practice cerebrospinal fluid is withdrawn before the serum injections are made. Even in cases in which some obstructive lesion already exists and no cerebrospinal fluid can be secured on lumbar puncture, it may still be safe to make the sub-

dural injection of the serum. It is only in rare instances in very young children, and in cases in which there is an obstacle to the inflow of the antiserum, that great caution must be exercised. Should unfavorable pressure symptoms arise, it is advisable to remove, by the usual procedure of lumbar puncture, or through suction by means of a syringe, part or all of the antiserum injected. A prompt relief of unpleasant symptoms due to pressure through this procedure has been noted by Brown.

The evidence at hand indicates that the earlier in the course of the disease the injections of the antiserum are made, the better are the results. A safe rule is to inject the antiserum at once, if a suspicion of meningococcal meningitis exists, without waiting to complete the bacteriological diagnosis by film preparation or culture. The occurrence of a turbid or purulent fluid at lumbar puncture is always suspicious and should call for immediate injection of the antiserum. During the prevalence of epidemics of meningitis, and perhaps at other times, cases of doubtful nature present themselves in which an exploratory lumbar puncture will show sometimes clear and sometimes turbid fluid containing the meningococci and possibly very few leucocytes. Immediate serum injections in these early cases often cut short the disease, as has been observed by Bradley and by Netter. Considerable emphasis should be placed upon the carrying out, more frequently than has been done in the past, of exploratory lumbar puncture for the diagnosis of epidemic meningitis. Increasing experience has shown that it is not safe to rely upon the gross characters of the cerebrospinal fluid to indicate the existence or non-existence of infection and inflammation of the meninges, and of its probable nature. The recent observations just alluded to leave no doubt that in the earliest stages of epidemic meningitis a clear fluid may be obtained, although at a period twenty-four to forty-eight hours later in the same cases the fluid may have become turbid or even purulent. Every effort should be made to carry out the serum injections in these early stages of the infection, since the experience at hand encourages the view that many, if not all, of the cases may at this stage be aborted.

The antiserum should be kept in the refrigerator until it is to be used and it should be warmed to the body temperature before it is injected. The minimal dose for a single injection may be

regarded as 15 to 45 c.c., except in young infants, in which smaller doses may be used with equal effects. As a rule, the serum passes readily into the spinal membranes when injected with a syringe or by means of gravity through a funnel and rubber tube attached to the puncture needle. It is desirable to remove as much cerebrospinal fluid as possible before introducing the serum, in order first to make space for the serum and to avoid unnecessary dilution of it, and next to facilitate its passage quickly into the ventricles and subarachnoid space of the brain. The spread of the antiserum into the brain is also favored by raising the foot of the bed after the injection is completed.

It is usually necessary to give more than one injection of the antiserum. Indeed, it sometimes happens that after the first injection there occur rapid fall in temperature, improvement of the subjective and other symptoms, and change from a state of unconsciousness to one of consciousness in twelve to twenty-four hours, and this continues without interruption until complete recovery. However, the rule is that repeated doses are required to bring about these conditions or to establish them on a permanent basis. Experience indicates that the disease can be shortened and the liability to relapse lessened by repeating the injections several times. For the present, the arbitrary plan of a daily injection for three or four days has been adopted, but it is imperative to control the injections by observations on the changes in the appearance and nature of the cerebrospinal fluid, and particularly upon whether it becomes clearer day by day and the meningococci grow fewer or disappear altogether. As long as the microscope shows the diplococci to be present, the injections should be repeated daily. As is obvious from what has already been stated concerning the viability of the diplococci in cases treated with the antiserum, they are to be searched for in film preparations where they may still be found when they are incapable of growth in cultures. No danger is to be apprehended from the repetition of the injections of the antiserum, although urticaria is not infrequently produced. The severer symptoms of anaphylaxis can, it appears, be disregarded. *In the fulminant and in certain very severe cases of the disease, it is advisable to make two injections of maximal doses of the antiserum within the first twenty-four hours, and possibly even in the next twenty-four-hour*

period. By means of estimating quantitatively the antiproteolytic ferment in the antiserum, Dochez has determined that the injected serum tends to leave the meninges within about twenty-four hours of the time of its introduction. In view of the manner of action of the antiserum and its rapid passage from the cerebrospinal fluid into the blood, it is obvious why the injections must be repeated several times.

A certain but apparently not very large number of cases resistant to the antiserum have occurred. This resistance declares itself in the failure of the diplococci to be controlled in an equal degree or at all in their cultural properties by the serum injections. Corresponding with this condition, the changes of the favorable nature described in the exudate fail also to set in, so that the progress both of the infection and the inflammation may not be arrested. These cases may either ultimately respond to the injections or they may proceed to a fatal termination either while the disease is still in the acute or subacute stage, or after it has become chronic. Some of these resistant cases certainly respond ultimately to the injections. In one such case, Fulton injected a total of 650 c.c., and in another Netter injected a total of 823 c.c. of the antiserum, both eventually making complete recoveries. In the case of Netter twenty-two separate subdural injections of the antiserum were made. We cannot as yet explain these resistant examples of the infection. Whether they depend upon differences in the resistance of the diplococci, or some peculiarity of the host, is not known. Certain differences in the readiness with which different strains of the diplococci undergo disintegration in leucocytes has been observed, but no exact comparative studies have yet been made which show that the diplococci cultivated from the cases not responding to the serum are relatively more resistant to other injurious influences. Since the degree of concentration of the antiserum in the subdural space is a matter of such moment in respect to the inhibition of the multiplication of the diplococci, it would seem advisable in this class of cases to employ a few maximal doses, perhaps 60 c.c. or more at one period in the course of the disease, in the hope of interrupting the growth of the meningococcus.

The chronic forms and stages of the infection are the least promising, as might be expected, and yet not a few such cases, in

which the antiserum was employed very late and after the disease had lasted many weeks, have been brought to rapid recovery. The chronic cases still showing febrile temperature and meningococci in the exudate are the more favorable, while the afrebrile ones in which the diplococci cannot be found or in which hydrocephalus is already well established appear to be far less promising. It has been remarked by Robb particularly that the chronic types of the disease are greatly diminished in hospitals as a result of the employment of the serum in all cases entering them. This change in the types of the disease is effected by the greater number as well as rapidity of the recoveries among the infected, thus eliminating many of the persistant, progressive, and more chronic types of the malady. When the serum-treated cases terminate fatally they usually do so because of the great severity of the initial infection, or as a result of some secondary pathological process or complication and chiefly during the early period of the disease. It happens sometimes, in young children especially, but also in adults, that an infected exudate becomes locked up in the cerebral ventricles and little or no fluid is contained in the spinal subdural space. The injections of the antiserum by lumbar puncture in such cases is unsatisfactory. The amount, relatively, of the serum which can be introduced without encountering a severe obstacle is not great, and although it may exert its favorable effects on the exudate present about the spinal cord it cannot reach the exudate about the brain except through the medium of the general circulation, which we have found a totally inadequate means. In order to obviate this drawback, Cushing and Sladen have found it practicable to drain the cerebral ventricles and to inject the antiserum directly into them. In very young subjects the injection can be made through the fontanelle, in older subjects trephining is required. Fischer and Netter have also carried out the intraventricular form of injection of the antiserum, at least up to the present time without doing injury, but thus far also without strikingly favorable results.

A certain but as yet moderate number of cases of the disease apparently recovering after the serum injections suffer a relapse. This number has grown smaller since the introduction of the method of consecutive serum injections. The relapses should be treated as was the original infection, *i.e.*, the consecutive injections of the

antiserum should be resumed immediately upon recognition of the relapse. In certain instances the recognition of the relapse is not easy. Some patients who are improving but have not yet become afebrile, and in whom the other symptoms have not yet abated, suffer a change for the worse. The circulating leucocytes may rise in number sharply. The cerebrospinal fluid shows increased turbidity and evidence of diplococcic multiplication. In a second series of cases the relapse is more definite, since the patients may have been free of fever and other severe symptoms for days, when the relapse is ushered in by rise of temperature, the reappearance of other symptoms, and the changes mentioned in the blood leucocytes and in the cerebral exudate. On the whole, mere rise of temperature alone is a less serious, as well as a less certain indication of relapse, than the return of mental symptoms. In a very small number of instances of recovery under the serum treatment, the relapse has occurred after an interval of several weeks. The late relapse may be favored by traumatism, as in a case observed in the Philadelphia Municipal Hospital, or may appear without any recognized contributing cause. The treatment of the relapses, when promptly begun and actively carried out, has been quite as satisfactory as the treatment of the original infection.

In this country, we speak less of the posterior basic form of diplococcal meningitis than they do in Great Britain, and we are inclined to view that form of meningitis not as being a separate entity, but merely as representing a late stage, and possibly a particular form of localization of the same infection which produces epidemic meningitis. Some difference of opinion exists among bacteriologists as to whether the diplococcus present in the cerebrospinal meninges in posterior basic meningitis is the same as that occurring in epidemic meningitis. The study by Wollstein, under my direction, of several different strains of the diplococcus obtained from typical cases of basic meningitis failed to exhibit any characters either cultural or pathogenetic which separated the two groups of meningococci. It may, therefore, be expected that the serum treatment of cases of posterior basic meningitis will be as successful as the same treatment is of a corresponding series of cases of undoubted epidemic meningitis. Since the posterior basic cases

usually come under observation relatively late in the course of the infection, they will probably yield to the treatment with difficulty, just as the chronic stages of the usual type of infection are favorably influenced less readily than the more acute ones. Moreover, a peculiarity of the basic forms of meningitis is the associated hydrocephalus, which is itself a menace. It may be that the combined injections of the serum by lumbar and intraventricular punctures may yield better results.

A certain although not large number of cases of associated tubercular and meningococcal meningitis have been described by several observers. Apparently the tubercular infection is the primary one upon which the diplococcus becomes engrafted, the latter being possibly the immediate cause of death. These cases present certain important peculiarities which should be mentioned. The cerebrospinal fluid may be clear or turbid. Both polynuclear leucocytes and lymphocytes tend to be increased. The film preparation, if stained with methylene blue or by Gram's method with gentian-violet and safranin, will show only the diplococci. Unless, therefore, attention has been arrested by the excessive lymphocytes, the occurrence of tuberculous lesions elsewhere in the body or the atypical course of the malady, tubercle bacilli may not be sought. These cases, as would be expected, are not influenced favorably by the serum treatment. This form of mixed infection occurs especially among children. Netter has recorded several such among his series of serum-treated cases, and Marsh and Williams have described in detail four cases of this form of mixed infection in three of which during life the tubercle bacillus and the meningococcus were demonstrated, and in the fourth the tuberculous lesion was established by autopsy.

RESULTS OF THE SERUM TREATMENT

In estimating the results of the serum treatment of epidemic meningitis we possess two sets of criteria: first, the statistical facts of mortality, and second, the influence on the course of the disease and the sequelæ. Both sets of criteria should be examined. In making this examination we can eliminate from all consideration the cases which were treated by subcutaneous injections, since the

consensus of opinion is to the effect that no appreciable influence is exercised on the course and termination of the disease by that mode of treatment.

Confining our attention to the records of cases treated by the subdural method of injection of the antiserum, it can be affirmed that the course of the disease is shortened, the severity lessened, the recovery made more perfect so that the sequels are few, and the fatalities reduced. That the course should be shortened is to be expected by reason of the restraint which the serum exerts on the free multiplication of the meningococci and the corresponding cessation of exudation of leucocytes. Of 350 cases of meningitis which recovered during the recent epidemic in New York before the serum was introduced, the disease lasted in 3 per cent. one week or less, and in 50 per cent. five weeks or longer (Holt). In 228 cases treated by the antiserum, analyzed by Flexner, the duration of active symptoms after the first injection was approximately eleven days. This period does not include the time required for the disappearance of the rigidity of the neck and the Kernig sign, which often persists several days or one or more weeks after all active symptoms of the infection have disappeared.

Connected with the average duration of the disease is the manner of its termination. Epidemic meningitis rarely, if ever under ordinary circumstances and under the previous methods of treatment, terminated by crisis. The rule is for the symptoms gradually to subside. Of 273 cases treated with the serum, in which the histories permitted of a conclusion as to the manner of termination of the disease, analyzed by Flexner, in 200 the symptoms disappeared by lysis and in 73 by crisis, whence it has been concluded that an abrupt or a critical cessation of symptoms in serum-treated cases may be expected in 25 per cent. of the patients who recover. The permanent sequelæ noted among the serum-treated cases have been very few excepting deafness, which has been noted a number of times, and there have scarcely been others. The deafness has in most but not in all instances been noted before the serum injections were begun. It is at least probable that earlier injections of the serum may diminish this sequel. Instances of mental impairment, paralysis and blindness have been almost entirely absent.

Turning now to the statistical considerations, it will be instruc-

tive to introduce a tabulation of a series of cases which have been serum-treated, and to compare their fatalities with those among cases occurring in the same place and time and non-serum treated.

TABLE I.

Reported series of cases treated with the antimeningitis serum and the mortality among non-serum-treated cases.

By whom reported.	Number of serum-treated cases.	Per cent. mortality.	Per cent. of mortality of cases non-serum treated.
Dunn*	40	22.5	70
Chase and Hunt*	12	25	90
Sladen*	23	13	64
Fulton*	22	31	78.4
Koplik*	15	13.3	60
Ladd*	31	35.5	?
Morgan and Wilkinson*	10	30	?
Robb*	90	30	75
Ker*	30	43	80
Krohne†	59	40.6	66
Schoene‡	30	27	53
Levy†	23	21.7	78
Többen†	29	34	56
Netter*	50	18	83

* Flexner serum employed. † Wassermann serum employed. ‡ Jochmann serum employed.

The largest single series of serum-treated cases subjected to analysis is that of Flexner, and it contains the composite results obtained from the employment of the antimeningitis serum prepared at the Rockefeller Institute, New York. In all there are 712 cases considered in the analysis. The cases occurred in the United States and Canada, Great Britain and France. There were 488 recoveries and 224 deaths, which gave a mortality of 31.4 per cent. The facts relating to this series of cases are presented in two tables, in the first of which the cases are subdivided according to certain age periods, and in the second the total cases of each age period are further subdivided according as the serum was injected in the three arbitrarily chosen periods of duration of the disease.

Table II shows that the highest mortality occurred in the first two years of life, but contrary to the rule under the older forms of treatment, in which the mortality was 90 per cent. or over, in this series it was 42.3 per cent. The second age period is from 2 to 5 years, in which the mortality was 26.7 per cent. The third age period embraces children from 5 to 10 years of age and gave the

lowest mortality of all, viz., 15.9 per cent. The next period extends from 10 to 15 years and gave a mortality of 27.7 per cent. The next period of from 15 to 20 years showed a considerable rise in mortality, equaling 32.7 per cent., and the last period, embracing the cases of 20 years and over, gave the next highest mortality of all, viz., 39.4 per cent.

TABLE II.

Cases of epidemic cerebrospinal meningitis treated with the antimeningitis serum; cases analyzed according to age groups.

Age.	Number of cases.	Recovered.	Died.	Per cent. mortality.
1 to 2 years	104	60	44	42.3
2 to 5 years	112	82	30	26.7
5 to 10 years	113	95	18	15.9
10 to 15 years	101	73	28	27.7
15 to 20 years	107	72	35	32.7
20 years and over	175	106	69	39.4
Totals	712	488	224	31.4

The only percentage of mortality among these cases which calls for somewhat further discussion is the one relating to the period of 20 years and over, since the one there given is so much higher than those of the earlier periods excepting the first, and approaches more nearly the average previous fatalities. This apparent discrepancy is not readily wholly accounted for at present. The explanations which suggest themselves are that among older individuals there tends to be a larger number of very severe, rapidly fatal or fulminating cases of the disease, or, that older persons are less subject to the beneficial action of the serum. Perhaps some light may be shed on this question by the examination of the results of the serum treatment of cases in this period by those whose experience with the use of the antiserum has been great enough to make it probable that they would obtain the maximum advantages which the serum has to offer. I have, therefore, tabulated the cases going into this period, contained in the reports of Dr. Robb, of Belfast, Professor Netter, of Paris, and Drs. Ladd, Dunn, Sladen, and Fulton in this country. The reports furnished by these physicians contain the records of sixty cases occurring among adults of 20 years and over, of which 42 recovered and 18 died, the gross mortality, therefore,

being 30 per cent. The largest single series of cases of this period reported were treated by Dr. Robb. He reports 24 cases of which 20 recovered and 4 died, giving a mortality of 17 per cent. Of the 5 cases of this period occurring in the recent epidemic in Paris, reported by Professor Netter as treated with the antiserum, all recovered. It would, therefore, appear probable that as experience in the use of the antiserum increases, the relatively greater mortality of the adult period will be diminished, and that, on the whole, the cases in adults would not appear to be influenced less favorably by the antiserum than those occurring among younger individuals.

In discussing the manner of the administration of the serum, the point was made that a strong effort should always be made to inject the serum as early as possible in the course of the infection. That a considerable difference in the results is produced by this practice can be shown most convincingly by means of a brief analysis of the mortality figures, arranged according to an arbitrary scheme, in which the periods of injections of the antiserum are divided into three groups.

TABLE III.

Cases of epidemic cerebrospinal meningitis treated with the antimeningitis serum; cases analyzed according to day of injection.

Ages at period of injection.	First to third day.			Fourth to seventh day.			Later than seventh day		
	Recvd.	Died.	Per cent. mortality.	Recvd.	Died.	Per cent. mortality.	Recvd.	Died.	Per cent. mortality.
1 to 2 years.....	16	1	5.8	22	10	31.2	22	33	60.0
2 to 5 years.....	24	6	20.0	40	12	23.0	18	12	40.0
5 to 10 years.....	43	8	15.6	35	6	14.6	17	4	19.0
10 to 15 years.....	36	8	19.0	23	9	28.1	14	11	40.0
15 to 20 years.....	25	17	40.4	25	8	24.2	22	10	31.2
20 years and over..	36	21	36.8	34	24	41.3	36	24	40.0
Totals.....	180	61	25.3	179	69	27.8	129	94	42.1

In the first group there have been placed all cases in which the injection was made from the first to the third day of the illness, as far as could be determined clinically. In the second group are all cases injected from the fourth to the seventh day of illness; and in the third, all cases injected later than the seventh day of illness. An examination of Table III is particularly instructive in bringing out the fact of the importance of early injections of the serum. The

results in the first two years of life are especially noteworthy. The extraordinary figures given under the first period of injection, in children under two years of age, in which of 17 cases one only died, can hardly be maintained, but the influence of the period of injection is shown by the rapid rise in mortality in the subsequent two groups. It chances that the total number of cases in each group among the 712 cases analyzed is so nearly equal that they are properly subject to comparison. Thus among 180 cases injected in the first three days the mortality was 25.3 per cent., among 179 injected from the fourth to the seventh day the mortality was 27.8 per cent., and among 129 cases injected at a later period than the seventh day the mortality was 42.1 per cent. It will be of some interest to introduce here a word concerning the next largest single series of cases, treated with a particular preparation of the antiserum, which have been brought together. Stimulated by the promising results obtained in the serum treatment of epidemic meningitis in this country and in Germany, Dopter undertook the preparation of a similar antiserum in France. He has reported briefly upon 196 cases treated with his preparation of the antiserum during the recent epidemic in Paris, which gave a total mortality of 15.86 per cent. This result is to be compared with the figures given in the statistics of Netter who employed the serum prepared at the Rockefeller Institute and who reported a mortality of 18 per cent. among serum-treated cases and 83 per cent. among cases occurring in Paris at the same time and treated by other methods.

In view of the various considerations presented, the conclusion may be drawn that the antimeningitis serum, when used by the subdural method of injection in suitable doses and at proper intervals, is capable of reducing the period of illness; of preventing, in large measure, the chronic lesions and types of the infection; of bringing about complete restoration of health, in all but a very small number of the recovered, thus lessening the serious, deforming and permanent consequences of meningitis; and of greatly diminishing the fatalities due to the disease.

BIBLIOGRAPHY

Bovaird: Meningococcus Septicemia with Sterile Cerebrospinal Fluid; Iridocyclitis; Flexner's Serum; Recovery, Arch. of In. Med., 1909, iii, 267.

Chase and Hunt: Serotherapy of Epidemic Cerebrospinal Meningitis, Arch. of Int. Med., 1908, i, 294.

Cushing and Sladen: Obstructive Hydrocephalus following Cerebrospinal Meningitis, with Intraventricular Injection of Antimeningitis Serum (Flexner), Journal of Experimental Medicine, 1908, x, 548.

Dochez: Proteolytic Enzymes and Antienzymes of Normal and Pathological Cerebrospinal Fluid, Jour. of Exper. Med., 1909, xi, 718.

Dopter: La sérothérapie antimeningococcique dans 196 cas de méningite cérébrospinale épidemique, bull. et mémoires de la soc. méd. des hôpitaux de Paris, 1909, xxviii, 39.

Dunn: The Serum Treatment of Epidemic Cerebrospinal Meningitis, Jour. of the Amer. Med Assoc., 1908, li, 15; The Method of Administering Antimeningitis Serum, Boston Med. and Surg. Jour. 1908, clix, 743.

Flexner: Experimentelle Cerebrospinal Meningitis und ihre Serumbehandlung, Centralbl. f. Bakt., orig., 1906, xliii, 99; Experimental Cerebrospinal Meningitis and its Serum Treatment, Jour. of the Amer. Med. Assoc., 1906, xlvii, 560; Contributions to the Biology of Diplococcus Intracellularis, Journal of Experimental Medicine, 1907, ix, 105; Experimental Cerebrospinal Meningitis in Monkeys, *idem*, 142; Concerning a Serum Therapy for Experimental Infections with Diplococcus Intracellularis, Jour. of Exp. Med., 1907, ix, 168; The Present Status of the Serum Treatment of Cerebrospinal Meningitis, Jour. of the Amer. Med. Assoc., 1909, liii, 1443.

Flexner and Jobling: The Serum Treatment of Epidemic Cerebrospinal Meningitis, Jour. of Exp. Med., 1908, x, 141; Analysis of 400 Cases of Epidemic Meningitis Treated with the Antimeningitis Serum, Jour. of Exp. Med., 1908, x, 690; Kurzer Bericht über 400 Fälle von Meningitis, behandelt mit intraspinaler Einspritzung von einem Antiserum, Centralb. f. innere Medizin, 1908, xxix, 893.

Fulton: The Serum Treatment of Epidemic Cerebrospinal Meningitis, with a Report of 22 Cases, Boston Med. and Surg. Jour., 1908, clix, 537, 572, 617.

Holt: The Treatment of Cerebrospinal Meningitis by the Serum of Flexner and Jobling, British Medical Journal, 1908, ii, 1336.

Jobling: Standardization of the Antimeningitis Serum, Jour. of Exp. Med., 1909, xi, 614.

Jochmann: Versuche zur Serodiagnostik u. Serotherapie der epidemischen Genickstarre, Deutsche med. Woch., 1906, xxxii, 788.

Ker: The Treatment of Cerebrospinal Meningitis with Flexner's Serum, Edinburgh Med. Jour., 1908, i, 306; also British Med. Jour., 1908, ii, 1340.

Kolle and Wassermann: Versuche zur Gewinnung u. Wertbestimmung eines meningokokken Serums, Deut. med. Woch., 1906, xxxii, 609.

Koplik: The Serum Treatment and the Prognosis under Various Forms of Therapy of Cerebrospinal Fever, Medical Record, New York, 1908, lxxiv, 557.

Krohne: Ueber die bisherigen Erfolge der Behandlung der epidemischen Genickstarre mit Genickstarre-Heilserum im Regierungsbezirk Duesseldorf, Zeitschr. für Medizinalbeamte, 1908, xxi, 78.

Ladd: Serum Treatment of Epidemic Cerebrospinal Menigitis, Jour. of the Amer. Med. Assoc., 1908, li, 1315.

Levy: Erfahrungen mit Kolle-Wassermanschen meningokokken Heilserum bei 23 Genickstarrekranken, klinisches Jahrbuch, 1907–08, xviii, 317; Bemer-

kungen zur Technik der intralumbalen Anwendung des meningokokken Heilserums (Kolle-Wassermann), Medizinische Klinik, 1908, iv, 1535, 1574.

Marsh and Williams: The Treatment of Cerebrospinal Meningitis in Children with Flexner and Jobling's Antiserum, The Liverpool Medico-Chirurgical Journal, 1909, xxix, 270.

Morgan and Wilkinson: Report of Ten Cases of Epidemic Cerebrospinal Meningitis Treated with Antimeningitis Serum, Archives of Internal Medicine, 1908, ii, 253.

Netter: Sur la méningite cérébro-spinale, etiolgie, prophylaxie, sérotherapie de la méningite cérébro-spinale, bull. de l'Acad. de méd., séance du 4 Mai, 1909; Septicémie méningococcique, sans méningite; éfficacité du serum anti-méningococcique, bull. de l'Acad. de méd., séance du 27 Juillet, 1909; Résultat du traitement sérothérapique dans soixantehuit cas de méningite cérébro-spinale; Un cas de méningite ayant nécessite vingt-deux injections de serum, *idem;* Liquide céphalo rachidien limpide au cours des méningites cérébrospinales (Première note: Liquide clair pendant les 24 premières heures de la maladie), comp. rend. des séances de la soc. de biologie, 1909, lxvi, 866.

Neufeld: Ueber die Wirkungsweise und die Wertbestimmung des Genickstarre Serums, med. Klin., 1908, ii, 1341.

Robb: The Treatment of Epidemic Cerebrospinal Fever by Intraspinal Injections of Flexner and Jobling's Antimeningitis Serum, Brit. Med. Jour., 1908, i, 382; also 1908, ii, 1341.

Schoene: Ueberblick ueber die Behandlung von 30 Genickstarrekranken mit Jochmannschen Meningokokkenserum, Die Therapie der Gegenwart, 1909, xlviii, 52.

Sladen: The Serum Treatment of Meningitis, Jour. of the Amer. Med. Assoc., 1909, li, 1318.

Többen: Zur Therapie der Meningitis cerebrospinalis epidemica, Münch. med. Wochenschr., 1907, liv, 2420.

Warfield: Acute ulcerative endocarditis caused by the meningococcus (Weichselbaum), U. of P. Med. Bull., 1903–4, xvi, 180.

Wassermann: Ueber die bisherigen Erfahrungen mit dem meningokokken Heilserum bei Genickstarrekranken, Deutsch. med. Woch., 1907, xxxiii, 1585.

Weichselbaum and Ghon: Der Microkokkus meningitidis cerebrospinalis als Erreger von Endokarditis sowie sein Vorkommen in der Nasenhöhle Gesunder und Kranken, Wiener klin. Woch., 1905, xviii, 625.

TREATMENT OF CANCER BY FULGURATION

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In 1907 Dr. De Keating-Hart reported to the Academy of Medicine an electrosurgical method for treating malignant tumors to which he had given the name of fulguration.

The application of this method, as used by Keating-Hart at the present time, comprises two separate stages: (1) a surgical stage in which the operator does his utmost to remove the tumor completely, or at any rate up to its accessible limits; (2) an electric stage, the fulguration properly so called, which consists in projecting on the operative field a shower of sparks supplied by an alternating current of high frequency and high tension.

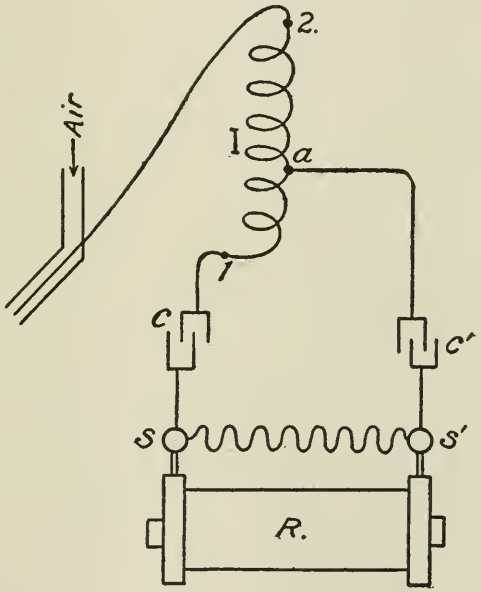
In the early days of the method the surgical portion of this process was of lesser importance; the knife was merely used to clear the way, and the tumor was removed in pieces if this manœuvre facilitated the intervention; but riper experience has shown that the preliminary surgical part is of primary importance. In his last report to the French Association for the Study of Cancer Keating-Hart says that the requisite minimum of the surgical portion is complete removal of the macroscopic lesions; he still approves of removal of the tumor in pieces, but this is a method of procedure not to be employed, in my opinion, except when unavoidable.

Fulguration itself has as its *immediate* aim the destruction of the fragments of tumor that have been left by the surgeon, either knowingly or unknowingly; its *ultimate* but principal aim is to set up a lasting reaction of the neighboring tissues, wherein consists the therapeutic value of the method.

The technic of the operation can be explained in a few words (Fig. 1). The high-frequency current escapes at the free end, 2, of the coil of an Oudin resounder *I*, which is an integral portion of a high-frequency generating apparatus. The following is the way in which a machine of this sort is arranged: suppose a Rhumkorff

coil, *R*, giving a spark of about 40 centimetres, and fitted with a high-speed interrupter. Each one of the poles of the secondary current, *S* and *S'*, is connected with a solenoid *I* by means of a condensor *C* and *C'*. But whereas the pole *S*, for instance, is invariably connected with the extremity 1 of the solenoid, the pole *S'* is connected with one of the coils of the solenoid at a point *a*, which can be moved nearer to or farther from the extremity 2. When sparks are produced between the poles *S* and *S'*, a current is generated in the circuit *S C* 1 *a C' S'*, and in its prolongation, *a* 2,

Fig. 1.



an alternating current whose periods rise to millions per second and whose tension reaches two or three hundred thousand volts. This current is led from the resounder to the patient by means of a wire ending in an electrode, the whole sufficiently isolated for the operator to be able to work without danger.

The patient is placed on a wooden table, that is to say, on a poor conductor; when under these conditions the electrode is brought near to the operative field, a series of sparks is emitted whose length can be regulated at will according to the effect desired.

Keating-Hart considers it very important that the heating

action of the sparks should be avoided; he has therefore devised special electrodes to accomplish this purpose. The metallic electrode is fitted in the axis of an isolating tube through which passes a steady stream of cold, filtered, and sterilized air or of some inert gas. Sterilization of the gas projected on the wound is indispensable in order to guard against infection. Fulguration is a painful process, and therefore requires either local or general anæsthesia. In fact, general anæsthesia is almost always necessary, and should be obtained by means of chloroform, to the exclusion of either chloride of ethyl, or of any mixture whose vapors might catch fire from the electric sparks.

While the surgeon keeps the wound wide open with metallic forceps held in his bare hands, the electrician carries the sparks over the entire raw surface, into all its corners, and over the skin around it. This manœuvre must be scrupulously aseptic. Since at the present time we have no apparatus that enables us to measure with precision the frequency, intensity, and tension of the current used, empiricism is our only guide. Experience has taught the operator what length of spark is most suitable for use, and during what length of time a certain point should be treated in order to obtain a therapeutic result without producing too marked destructive effects, which, in certain cases, might have serious consequences, as, for instance, in the viscera, veins, and arteries.

When the fulguration is finished the surgeon completes the operation; he endeavors to close up the loss of tissue as far as practicable allowing at the same time free drainage. During this last stage none of the instruments used for the initial removal of the tumor should be employed, and the hands of the operator and assistants should be cleaned afresh. Finally, the wound should be covered with a protective dressing that is very absorbent.

This being the principle of the method, let us see what are its results.

1. Fulguration produces immediate cessation of surface hemorrhage through vasoconstriction, and this hæmostasis is definite, thanks to the coagulation of the blood in the capillary vessels.

2. A few hours after the fulguration a serous exudation sets in, sometimes so profuse that the dressing is soaked. This liquid, rich in polynuclear leucocytes, seems to have toxic and perhaps cytolytic

properties, and when it stagnates in a wound that is closed or badly drained, it gives rise to symptoms of intoxication, with enormous elevation of temperature, capable of causing death. Still, we have yet to distinguish the part played in such accidents by the specific toxicity of the exuded serum and by the usual infectious agents.

3. The fulgurated tissues are mortified to a slight depth.

4. When this layer of dead tissue comes away, at the end of a few days, very active granulations are found proliferating underneath; a loss of tissue of this kind is usually repaired much more rapidly than a wound of similar extent that has not been fulgurated.

We can mention, in addition, that fulguration has an immediate anæsthetizing effect; this has been mentioned in a great number of cases, and I personally had a patient under treatment with whom the suffering disappeared altogether, although up to that time it had been incessant.

Microscopical analysis and experimentation on normal and pathological tissues enable us to see more intimately into the mechanism of the phenomena observed. A large number of experiments have been carried out of late, in Germany by Czerny, Wasielewski and Hirschfeld, in France by Manté, and especially by Berjouié and Triboudean, and in Belgium by De Nobele and Tytgatt. The results published do not altogether agree with each other, but a certain number of interesting facts seem to have been demonstrated.

Berjouié and Triboudean have studied the action of short and rapid sparks on the testicle of the white rat and on the liver and kidney of the rabbit, protecting these structures carefully from infection which disturbs the process of fulguration. Several of their conclusions are worthy of note and comment.

" Fulguration has an elective, destructive action on epithelial cells, while respecting connective tissue," within the limits of the intensities used; " it destroys all epithelial cells, without distinction of form and activity." To this has been added that it shows no special predilection for tumor cells.

The destructive effect of fulguration has no period of latency and is quite superficial; what it destroys it destroys from the start, but this layer is only a few millimetres in thickness.

" The line of suppuration between the dead and living portion

is quite distinct; the epithelial cells of the living tissue not only show no change but are in no wise influenced either in evolution or in reproductive power." From this we can conclude that the destructive action of fulguration is purely local; it extends a little beyond that of the knife; a caustic applied to the wound would do as much. It is therefore comprehensible that the therapeutic effects of fulguration are proportionate to the extent of the surgical removal.

Even if we admit, then, the curative value of fulguration for limited cases, it is inefficacious as regards cure, in the strict sense of the word, both for generalized cancer and for cancer with deep prolongations that cannot be reached surgically. It would be a deception to propose fulguration as an alternative for the classical treatment, that is to say, free removal of the tumor. By giving the patients the hope of possible recovery at the price of a slight operation they are placed in a state of false security, and if the tumor is still at a stage at which it can be profitably removed, they are led to delay making a decision until an operation is no longer possible.

"Fulguration is rapidly followed by an enormous afflux of leucocytes to the tissues treated, due to diapedesis through the walls of the congested vessels, and lasting only a few days."

In experimenting on the liver of the rabbit Berjouié and Triboudean remark finally "that—in their main lines—the destruction as well as the repair of liver tissue are identical, in their histological manipulations, whether they are produced by fulguration (two minutes at one centimetre), monopolar electrolysis (30 seconds, ten to fifteen milliamperes), cauterization (puncture with a thermocautery), or crushing (forceps)." The high-frequency spark, then, acting on healthy tissue and on the animal, has no properties that are extraordinary or in any way new.

Such are, in brief, the clinical data that have been established by Keating-Hart and the other clinicians who have tested the method. The cases that they have had in hand have, as a general thing, been very bad ones that had been abandoned by surgeons as incurable; for this reason we find a large percentage of bad results in their series, for which it is not fair to hold the fulguration method responsible. Thus, in the last series published by Desplats, there were as many as seven such cases out of a total of eighteen.

The patients counted as cured are those with whom the wound healed up rapidly, and who are living in a satisfactory condition of general health either without apparent recurrence or with a recurrence of a very torpid nature.

Keating-Hart with great frankness speaks of "apparent cures without pretentions as to the future." Is it possible to speak of real cures, of cures without qualification? This is quite another matter.

Thus, the rapid cicatrization commonly observed after fulguration cannot be looked on as a criterion of recovery; for a surgical operation, or a treatment by radium, gives similar cicatrizing results and long survival, sometimes even unhoped-for and inexplicable. Besides, cicatrization is not more common after fulguration than that which we obtain after incomplete removal and immediate reunion; but it is unquestionably more rapid than that which takes place when we leave the wound open. There is no question but that this is one of the strong points in the fulguration method.

Recurrence, that is mentioned in a certain number of cases, is relatively tardy. We have above referred to the intense reaction of connective tissue to the stimulus of the sparks. In practice this manifests itself by a process of fibrous transformation; this dense tissue surrounds the fragments of epithelium that have remained, constitutes a barrier to their progression, and transforms, so to speak, the cancer treated into a scirrhous tumor. A similar process is observed in the radium treatment of cancer; but after fulguration it appears to be specially active and efficacious.

In order to be able to speak of real recoveries we ought to be in possession of a large number of cases with histological and clinical control, and particularly with the test of years. So many so-called curative treatments of cancer have been advanced with the very best faith, that it is impossible to avoid a feeling of scepticism in the presence of a new attempt. Criticism, no doubt, has no right to a greater degree of blindness than has faith; yet with such delicate questions nothing but absolutely demonstrative facts ought to count. It is, therefore, not possible at the present time to say that fulguration cures cancer; it is not even probable that it can do so in cases where surgery is powerless. This does not mean

that fulguration has no value whatever; as a palliative means the method deserves to be taken into consideration and utilized.

The point, then, to settle is what cases ought to be treated by fulguration, and in this respect cancer must be considered in its extension and in its localization.

In cases that are suitable for operation, that is, when the tumor can be removed surgically in a simple mass with its prolongations and with a certainty of passing outside the limits of the disease, fulguration adds nothing to the efficacy of the removal. Let us take as a concrete example a cancer of the breast in its early stages. If the operator, after having isolated the vessel and nerves of the axilla, removes in one mass the thoracic portion of the big pectoralis major and pectoralis minor, lymph-nodes, and axillary fat down to the muscles laid bare, the operation gives to the patient the maximum chance of recovery, operative cure is rapid and without incidents, and the functional results ought to be good. To fulgurate such a wound is nothing more than to remove a few additional millimetres of tissue, while preventing reunion by first intention, which would prolong without advantage the duration of the treatment.

The case is no longer the same in the less satisfactory instances in which the operator is not sure of removing the tumor *in toto,* without taking it out in fragments, without opening lymphatics, or without leaving diseased lymph-nodes; and also in instances that are manifestly bad (those which we look on as inoperable, that is to say, logically incurable by operation), where the surgeon is certain that he cannot possibly remove the whole of the tumor. In these two classes of cases it seems legitimate to combine the operative intervention with fulguration. In the present state of our knowledge it is not too much to hope that the connective-tissue reaction brought on by the sparks will prevent for at least a while the fatal relapse. The imporant point to know is whether the drawbacks and risks of fulguration are not out of proportion with the possible benefit to be obtained.

As regards these drawbacks and risks: fulguration lengthens the duration of an operation; the wound cannot be united, as the dead tissue has to be eliminated, and the toxic lymph must find a free exit. In many cases infection cannot be avoided. These are

unquestionable drawbacks, but of minor importance, and can therefore be set aside.

Several cases of death after fulguration have been published. Death can also occur during the operation. When the sparks are applied to the thorax they may stop respiration and consecutively the heart. In practice these accidents can be avoided by an operator properly versed in the technic. But fulguration of the pneumogastric nerve must be absolutely forbidden; and when the integrity of this nerve is doubtful, Keating-Hart recommends that it should be resected without hesitation rather than fulgurated.

Other cases of death have occurred a few hours after fulguration, but these have generally been preceded by extensive operations on cachectic patients. It is manifest that the electric trauma is partly responsible for such a fatal issue, but do we not see non-fulgurated patients also succumb after such interventions?

Death occurring later still seems to be the result of sepsis, retention of toxic products, or of complications foreign to fulguration; but if the operation is cleanly done, if we take care not to infect the wound by blowing dusty air on it in cooling the electrode, if we provide thorough drainage by leaving the wound open or by removing the sutures at the first alarm, we shall have every chance of escaping this class of disaster.

In a word, fulguration cannot be considered as a harmless treatment, but a careful technic will enable us to steer clear of fatal accidents, particularly if we are cautious in not operating indiscriminately on patients that are really doomed beforehand. In the interest of the method it is better to refuse all cases of generalized cancer in which the benefit to be expected is really insignificant.

As rgards indications, all localizations are not equally favorable for fulguration. External cancers, of the breast or face, for instance, are naturally the most suitable. The difficulties of access in visceral tumors must, however, not be looked on as an insurmountable obstacle. Thus, very satisfactory results have been spoken of in treating cancer of the rectum; and Marion recently showed before the Society of Surgery a remarkable case of inoperable cancer of the bladder in which, after a temporary cystostomy, Keating-Hart fulgurated the cancer, obtaining perfect cicatrization of the vesical mucous membrane. Attempts have also

been made on uterine cancer by the vagina, and even by the abdomen after removal, but these results have not been satisfactory.

It is evident that the last word has not yet been said concerning fulguration. Our present ideas can only be looked on as provisional and open to revision. But it certainly seems that Keating-Hart has enriched the palliative treatment of cancer, and that fulguration, performed according to a good technic and in properly chosen cases, has a right to occupy an honorable position by the side of, if not above, X-ray and radium treatments.

Until further notice the best treatment of cancer is early and free removal. *An early diagnosis, no hesitation, and no half measures*—such is the formula to remember, until medicine has discovered the serum or curative remedy for which we are all so earnestly seeking.

THE USE OF TUBERCULIN IN TREATMENT

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We are at present at the height of a revival of interest in tuberculin treatment. Notably in Germany does enthusiasm abound and to a less extent in this country and in England and France. We are not yet in a position to give a final judgment of its value, but certain impressions have become so firmly and so generally established that they demand serious consideration. No doubt enthusiasm has carried many to place too high their faith in its efficacy while others assume a calm scepticism and patiently wait for the tuberculin hope to die as nearly all other therapeutic hopes have. We know that tuberculin is not a cure for tuberculosis, but if it can assist us even to a limited extent in our efforts to help afflicted individuals we shall be grateful. It will never be a measure of such wide influence that it will assume prophylactic and preventive importance. It is in the consideration of the individual case and the benefit it may bring this particular individual that its value rests. This value, which time alone will show whether we are justified in assuming, has brought home the question of tuberculin treatment to every practitioner. He is eager to know what has been accomplished, what views prevail, how the treatment is carried out and whether the method will lend itself to his own manipulation. It is from these viewpoints that we wish to consider the subject.

Universal condemnation followed the use of tuberculin after Koch's [1] original communication in 1890. Large doses were given, reactions were expected and sought and in spite of Koch's explicit warning no heed was paid to the extent of the disease or to the condition of the patient. We now look with amazement and horror at the charts of the period when patients already suffering and ill unto death were subjected every day to intense rigors followed by high fever and the symptoms which perhaps only those who have had a severe tuberculin reaction can fully appreciate. The repulsion on the part of the profession was so intense that one prominent

member did not hesitate to declare that to subject a patient to the tuberculin treatment was cause sufficient for a civil suit. In spite of this feeling men of prominence, notably Goetsch, Petruschky, Trudeau [2] in this country, and Von Ruck, and later Moeller, Spengler [3] and others continued quietly to use tuberculin and by modifying the dose and the interval and by selecting suitable cases, they were soon able to show that when properly used tuberculin certainly does no harm and seems in many cases to be of decided benefit. Eleven years after Koch's original communication the first article in favor of a rational use of tuberculin was published by Goetsch. Since then innumerable contributions have appeared and for the most part favorable in their view of tuberculin. The reports of Trudeau, Sahli,[4] Denys,[5] Moeller, Petruschky, Löwenstein,[6] Bandelier and Roepke,[7] and Brown [8] are particularly noteworthy and should be consulted by all engaged in tuberculin treatment.

We have previously said that the main evidence we have for the value of tuberculin in treatment is the impression of the men who have used it. There is no convincing experimental evidence of its value and the available clinical statistics are open to serious criticism. Some measure of success has followed the numerous efforts to produce immunity to tuberculosis culminating in the brilliant work of Behring and Koch. When the disease, however, has once become established, control of its progress is impossible. All types and every conceivable modification of the tubercle bacillus and of allied organisms and their products have been used. The extravagant claims of one investigator are overthrown by the negative results of another and all that one can affirm with certainty is that in nearly every instance the treated animals have survived longer than the untreated controls. As disappointing as this lack of experimental evidence is, it by no means contradicts the moderate claims that are now advanced for tuberculin. The susceptibility of laboratory animals and the vast difference between naturally acquired and experimentally induced tuberculosis do not allow an exact comparison with the disease in man. The results do emphasize that we cannot expect too much from tuberculin, that it is certainly not a cure for tuberculosis, but they do not contradict that under some conditions it may have a beneficial influence upon the course of the disease.

From the clinical side it has been claimed that this beneficial influence is manifested in a more rapid and satisfactory improvement during treatment and that the improvement won is more enduring than in untreated patients. The number of cases reported by most observers has been too small to give their results much weight. Of the many who speak favorably and even enthusiastically of the value of tuberculin only a few have figures that lend support to their view. The statistics from Belzig published by Moeller, from Cottbus by Nagel, and from the Adirondack Cottage Sanatorium by Brown are the only available ones that are extensive and carefully enough compiled to be of value. The inherent difficulty of giving satisfactory tuberculosis statistics is particularly appreciable in this field. The diagnosis of incipient pulmonary tuberculoss when tubercle baclli are absent from the sputum is almost entirely a matter of individual skill and judgment. This personal equation makes it impossible satisfactorily to compare the results obtained by one observer with those of another. We are not surprised that some report 100 per cent. cures in closed and early pulmonary tuberculosis. If every patient who presents himself for examination and shows some trifling deviation from the normal physical pulmonary signs is immediately diagnosed tuberculosis, or if tuberculin is made the ultmate test of a correct diagnosis, similar results may be obtained with any or with no treatment. In the moderately advanced group the variation in the character of the cases from one just missing the incipient to one almost in the far advanced class, precludes almost any attempt at comparison. Even with the most honest effort it would be impossible to select two groups, each of 100 patients, that would allow comparable inferences about the value of two different methods of treatment unless one method were immeasurably superior to the other. We must admit that such statistics as we have are more suggestive than convincing, but their almost unanimous accord and particularly their agreement in many details give them unusual importance. The points they indicate are these,—the most successful immediate and lasting results are obtained among patients with slight and early lesions. As all early cases offer a good prognosis under proper treatment these results are not surprising. The difference between the results obtained with and without tuberculin treatment under

surroundings otherwise the same are by no means striking, although what difference there is falls in favor of tuberculin. The most conclusive results are obtained among the moderately advanced cases, those treated with tuberculin giving a much higher percentage of apparently cured and arrested cases. In the far advanced group there is the same difference in favor of tuberculin, although not as striking as in the moderately advanced class. When followed for many years patients who have received tuberculin maintain their improvement more satisfactorily than the untreated.

The impressions men who use tuberculin receive cannot be accepted as objective or scientific evidence. The room for error is large, but where such impressions are almost unanimous they do, it seems to us, acquire considerable importance. Until the number of treated cases becomes larger and statistics are more accurately gathered it must remain our chief assurance of the value of tuberculin treatment. Of the many who have used tuberculin we know of but two who have prominently denied it any value. The others are uniform in their estimation of its importance in selected cases at least. Cases of moderately advanced pulmonary tuberculosis who have remained stationary under the usual hygienic-dietetic régime and have then shown unexpected improvement with tuberculin are the common experience of all who use it. Such instances particularly have gained for it more and more adherents and have strengthened confidence in its value.

If tuberculin treatment has such value as we, with so many others, believe it to possess, it is important that it gain a still wider foothold. Until now used almost exclusively as an auxiliary in the sanatorium management of tuberculosis it is rapidly coming to demand a wider application. Tuberculin in no way changes our views regarding the importance of those measures spoken of as sanatorium treatment whether they are practiced in the home or in institutions, and every one is ready to concede that they can be more satisfactorily carried out in institutions than elsewhere. But not all patients, even not all favorable cases, can be admitted to sanatoria and those who have spent their allotted time there demand just as careful management after discharge as during their residence. From six months to a year is seldom long enough to make a cure lasting and sanatoria cannot keep patients until the result is

guaranteed. For tuberculin to be efficacious it must be administered over a long period of time. It is scarcely worth beginning the treatment if it cannot be continued for at least six months. There are then many patients who never enter sanatoria, who may be benefited by tuberculin, some who have started tuberculin while away and require its continuation after returning and others who come home from an institution improved or arrested, whose subsequent course may be favorably influenced by its use. The treatment of these patients falls in the hand of the general practitioner and even if he is unwilling to take the time and pains necessary to conduct successfully the tuberculin treatment he should be familiar with the views regarding it and the general principles underlying its use. Our work among out-patients at the Phipps Dispensary for Tuberculosis of the Johns Hopkins Hospital has convinced us that tuberculin treatment may be satisfactorily and successfully administered to ambulant patients even without interruption of their usual occupation. We appreciate that in the beginning our inexperience led us into many minor faults that with more practice might have been avoided, but with care and perseverance any one who has the necessary interest and patience may learn to conduct tuberculin treatment safely.

When first approaching the question one is bewildered by the large number of tuberculins or tubercle bacillus products that are offered to chose from. Each has its advocate and some pretended superiority over other forms. In general all the preparations may be divided into four groups.

First, the filtrates of liquid cultures of the tubercle bacillus. The most important members of this group are the "O. T." or original tuberculin of Koch and the "B. F." or broth filtrate of Denys. "B. F." is prepared by filtering mature cultures on glycerin bouillon first through filter paper and then through a Chamberland filter. It is a clear pale yellowish liquid of an agreeable pungent odor. A little carbolic acid and a little thymol are added as preservatives. "O. T." is prepared by reducing a six to eight weeks old glycerin-bouillon culture to one-tenth its volume at 90° C., adding 40 per cent. glycerine and filtering. It is a dark brown, rather viscid liquid of a similar but not so fragrant odor as "B. F."

The second group consists of suspensions of the ground-up portions of the tubercle bacilli themselves. The type of the group is Koch's tuberculin rest or "T. R." Young cultures dried in vacuum are pulverized in a mortar, shaken with water and the emulsion centrifugalized. The sediment after repeated washing and centrifugalization is suspended in a 20 per cent. glycerine solution, each c.c. of the emulsion containing 10 mg. of solid substance. Similar preparations are made by others from tubercle bacilli killed in various ways.

The third group consists of extracts of the bacilli. They have been treated with alcohol, with ether, with chloroform, with alkalies, with acids, etc. The most widely known preparation in America is Von Ruck's watery extract, and the most notorious the preparations of Von Behring—"Tulase" and "Tulaselaktin."

The fourth group consists of combinations of the foregoing varieties. The new tuberculin or bacillen emulsion of Koch, "B. E.", is the same as "T. R.", only it has not been extracted with water and centrifugalized so that portions of the culture fluid or "B. F." remain. Beranek's tuberculin, so highly prized by Sahli, belongs to this group.

Innumerable variations from these types have been produced, comprising changes in the culture fluid and in the method of treating the cultures. The most interesting variation, however, has been the source and character of the organism. Human, cattle, bird, and fish, tubercle bacilli have been used and the allied acid-fast group of Moeller. Interest has now narrowed down to tuberculins from the human and bovine type of the bacillus. Unless otherwise specified they are always made from the human type. In practice "O. T.", "B. F.", "T. R.", and "B. E." are the products that have met with most favor and are the ones almost universally selected. They stand in popularity approximately in the order given.

The mechanism of immunity in tuberculosis is still obscure and, although theoretical considerations may point to certain tuberculins as more desirable than others, there are no experimental or clinical grounds to support such contentions. Indeed from the evidence at hand we must conclude that the active principle in all is the same. From an unwarranted comparison with other diseases where the

conditions are better understood, it has become popular to speak of the antitoxic properties of the filtrates and the bactericidal properties of the bacillary bodies. It is important to emphasize that no such distinction exists. The tubercle bacillus produces no soluble toxin such as the diphtheria and tetanus bacilli do and the activity of the filtrates as well as of the emulsions depends upon the specific proteid of the tubercle bacillus itself. There may be differences in the degree and intensity but never in the quality of the reaction produced. Immunity to one form of tuberculin carries with it immunity to all other forms. Such differences as exist are largely physical. It is said that reactions come more unexpectedly and are more prolonged when bacillary emulsions are used than in treatment with the filtrates. This may be explained by the difficulty of getting uniform suspensions of tubercle bacilli or coarse particles of their ground-up bodies. It would seem, and perhaps for this reason as well as their more ready absorption, that treatment with the filtrates is more easily managed. It is advisable that beginners make at least their first trials with " O. T." or " B. F."

Following the more or less sharp distinction that has been drawn between the bovine and human types of the tubercle bacillus, interest has attached to the tuberculins from these two sources. Spengler was the first to call attention to what he considers differences in their action, claiming that tuberculin from bovine bacilli is more satisfactory in the treatment of infections with the human bacillus and tuberculin from human cultures in bovine infections. The truth of this contention is by no means established, but granting that it were, there would still be considerable difficulty in its proper application. How are we to know that a particular infection is due to the human or bovine type of bacillus? Cultivation of the organism from each case is impracticable. Spengler attempts to distinguish the two in the sputum by methods of staining, but his results have not been confirmed. Detre's differential cutaneous test has been shown to be unreliable. We are certainly no nearer accuracy if we follow Nathan Raw and call all gland, bone, serous membrane, and miliary tuberculosis, bovine infections and all chronic pulmonary tuberculosis, human infections. Such inconsequent classifications are no longer possible in view of the results of careful and painstaking cultural investigations. Lewis and

Duval have shown that glandular lesions are as commonly due to the human as to the bovine type of bacillus. It may be noted too that this view is directly opposed to the efficacy Wright attributes to treatment with homologous vaccines. Pottenger has offered some clinical evidence to support the justice of Spengler's claims. In a case of pulmonary and glandular tuberculosis he noted whenever human tuberculin was given that the glands improved while the pulmonary lesion grew worse and when bovine tuberculin was administered the glands markedly enlarged while the pulmonary lesion improved. Such a remarkable observation needs confirmation before it can be generally applicable. In our own experience we have been unable to see any such marked difference in the action of tuberculin from the two sources. Several cases that showed unusual sensitiveness to human tuberculin were put upon products from bovine bacilli in the hope that they would more readily tolerate it. However, no change in the susceptibility was noted. The immunization of calves with human bacilli is frequently quoted in support of the view, but it seems to us quite irrelevant in this connection.

Although the processes of immunization in tuberculosis are still but little understood it is important before beginning treatment to have in mind a clear idea of what one wishes to accomplish. Tuberculin acquires its characteristic toxicity only when an individual has become infected with tuberculosis. Healthy animals as well as healthy human beings stand large amounts without showing any reaction. Hamburger has given infants several hundred milligrammes without producing any symptoms of intoxication. When, however, the body harbors even a trivial tuberculous lesion a remarkable change occurs. The thousandth of a milligramme may then be highly toxic. Following its injection there is redness and swelling at the site of introduction, marked constitutional symptoms and an inflammatory reaction about the lesion. This altered relation to products of the tubercle bacillus in an infected animal was thoroughly discussed by Koch [1] in his original communication and was the basis of his therapeutic attempts with tuberculin. When a healthy guinea-pig is inoculated with a pure culture of tubercle bacilli the point of inoculation rapidly heals. After from ten to fourteen days a nodule forms which soon breaks

down, leaving an ulcer that persists to the time of death of the animal. If the inoculated animal has been previously infected with tuberculosis quite a different sequence of events occurs. After one or two days the point of inoculation is red and infiltrated and the area rapidly becomes necrotic, leaving a shallow ulcer that soon permanently heals without the adjacent lymph-glands becoming infected. The tuberculous animal then shows a remarkable sensitiveness towards the second injection of tubercle bacilli and a reaction follows that must be interpreted as a defensive mechanism, for superinfection is prevented. This ability of tuberculous animals to resist a second inoculation is still more strikingly illustrated in Roemer's experiments. Healthy and tuberculous guinea-pigs were inoculated with fatal doses of tubercle bacilli. The previously healthy animals rapidly succumbed to a generalized tuberculosis while the tuberculous animals suffered no appreciable effects from the inoculation, outliving the controls and ultimately dying as the result of the original infection. While the second injection had no power to stem the course of the disease already acquired, the animals were able completely to resist a renewed invasion, not even the regional lymph-glands becoming infected.

This sensitiveness for tuberculin is analogous to the general principle of anaphylaxis that has come to assume so prominent a rôle in our ideas of immunity. The same reaction the body is able to assume towards any foreign protein that may be introduced. An animal that has once received egg-white or horse-serum is made seriously ill by a second injection and killed if the amount be large enough. A mechanism that under many conditions may be protective is under other circumstances destructive and the animal really dies, as Vaughn expresses it, from over-protection. How this hypersensitiveness is developed is not altogether satisfactorily understood, but certain theoretical considerations correspond with the facts so far established. A certain length of time elapses between the primary injection and the development of hypersensitiveness, the height of the reaction being attained in from ten to fourteen days. During this interval the body presumably elaborates certain antagonistic substances which from analogy we assume to belong to the general group of antibodies. As albumen and other proteids are in themselves innocuous it is probable that the toxic

substance is liberated by disintegration of the proteid molecule. The exact nature of the antibodies is unknown, but they are usually designated as lysins or ferments. Certain evidence would indicate that they are free in the blood stream and others that the cells themselves have become highly sensitized. It has been supposed that the proteid upon its original introduction is so gradually disintegrated that no toxic symptoms arise. This activity, however, stimulates such an intense production of antibodies that, upon the second introduction of the proteid, disintegration proceeds so rapidly that the full toxic effects become manifest.

Under natural conditions in the tuberculous this hypersensitiveness to tuberculin shows great variations. While we know that it is diminished and frequently entirely absent in the final stages of the disease we can never postulate from clinical data what the relation will prove to be. Attempts to base prognostic inferences upon its intensity have been unsuccessful. Artificially we may to a great extent increase or decrease the sensitiveness of an infected individual to tuberculin. It is increased by the repeated injection of relatively large doses and particularly after an amount large enough to liberate a general reaction. Loewenstein was the first to show what an important part this increasing sensitiveness plays in the diagnostic use of tuberculin. An individual who fails to show any reaction to the first injection of 0.1 or 0.2 mg. of tuberculin may react violently upon the second, third, fourth or fifth repetition of the same dose. Occasionally it is possible to obtain a reaction even when the subsequent doses are diminished. Thus a patient failed to respond to 1 mg. then to 0.5 mg., but gave a sharp reaction to the third injection of 0.2 mg. It is important to emphasize that this increasing sensitiveness develops only in infected individuals. In the uninfected no number of repeated injections, no matter how small or how large they may be, will ever produce a reaction. The increased sensitiveness after a dose liberating a reaction has been emphasized by Koch. When a mild reaction occurs after a given dose a more severe one usually follows its repetition and indeed they may become more and more severe after each succeeding injection of the same dose. After such reactions patients will often show a marked sensitiveness to smaller doses which they had previously taken with impunity.

Sensitiveness to tuberculin may be diminished by the frequent repetition of doses just short of the amount necessary to produce a reaction, but far more successfully by the administration of gradually increasing doses. By the latter method susceptibility to tuberculin may be completely obliterated so that a patient who before treatment has reacted to the fraction of one milligramme may tolerate many grammes with no appreciable symptoms. This condition of acquired tolerance for tuberculin has been spoken of as tuberculin immunity. The mechanism of its development we cannot satisfactorily explain. From analogy with diphtheria toxin immunization it would seem probable that the body cells themselves do not become insensitive. Wasserman believes he has demonstrated by complement absorption experiments the presence of antituberculin in the blood of treated patients. While it is certain that this substance is not an antitoxin in the same sense as diphtheria antitoxin, it is possible that the toxic portion split off by the disintegration of tuberculin may find a binding substance in the blood. Wolff-Eisner suggest that certain tissues and organs may develop receptors which hold the tuberculin and prevent it reaching vital organs.

No doubt hypersensitiveness represents at least a protective attempt on the part of the infected body. Evidence indicates that it plays an important rôle in the prevention of reinfection and thus it may be of special advantage in the immunity acquired by an infection successfully overcome. There is, however, no reason to believe that it is similarly useful when the disease has once become established. To its presence are due those symptoms which are usually spoken of as toxic and the general nutritional disturbances induced may seriously impair the natural resources of the body for controlling the local disease. This at least is what clinical experience teaches us. As tuberculin tolerance is acquired there is usually a noteworthy change in the general condition of the patient. The appetite and digestion improve, energy and vigor increase, and nervous symptoms abate. It is reasonable enough to assume that the improved general condition will allow the natural defense resources of the body to come more satisfactorily into play. It is significant that with returning sensitiveness the usual symptoms of the disease again become prominent, to subside once more when tuberculin

immunity is re-established; that when relapses occur sensitiveness reappears; and that as a general rule in manifest tuberculous disease, when it is impossible to overcome the patient's hypersensitiveness and procure even a moderate measure of tolerance for tuberculin, improvement in the general and local conditions does not occur.

Sahli, Denys, and others, contend that this "giftfestigung" or toxin immunity is all that tuberculin treatment can accomplish. Certainly we have no satisfactory evidence that any degree of bacterial immunity is ever attained. It has long been known that the healing of a tuberculous focus under the influence of tuberculin treatment in no way differs from the healing by natural processes of repair which so frequently supervenes. It is thought that fibrosis may occur more rapidly and more thoroughly, but this is by no means satisfactorily established. The significance of agglutination and of phagocytosis is not fully understood. If they assist in the processes of control and healing, the exact part they play is unknown and their artificial stimulation cannot be successfully governed.

Wright has sought to bring scientific accuracy into tuberculin treatment by measuring the effect of each dose upon phagocytosis. While no doubt variation in the activity of the leucocytes occurs, the accuracy of his method of estimating the opsonic power of the serum is questionable. His observations have led him to rely upon the administration of small amounts of tuberculin frequently repeated, rather than to attempt a gradually increasing dosage. The results this method is claimed to have procured, especially in surgical forms of tuberculosis, are noteworthy even though we refuse to recognize the value of opsonic estimations. It would seem reasonable to assume that in certain instances where a sluggish tuberculous focus is so cut off from the general circulation that no products from the disease can be absorbed and so produce a protective response, small doses by stimulating hypersensitiveness might be of value. Such a condition would hardly be possible in pulmonary tuberculosis and seems to us to be of more theoretical than practical importance in other forms. It is in localized surgical tuberculosis, notably in skin tuberculosis, that sensitiveness to tuberculin is as a rule already most highly developed. Our own

experience in the treatment of a few instances of lupus, tuberculous glands, pelvic tuberculosis and corneal tuberculosis has led us to adhere, even in such cases, to an attempt at producing tuberculin immunity rather than to the continuous repetition of small doses. The relative value of these two methods is as yet by no means settled. It would seem that satisfactory results are obtained by either and we must await further knowledge and experience before reaching a definite conclusion.

It will be remembered that the reaction was an important part of the immunizing process first outlined by Koch and even in later publications he has never completely relinquished the idea of its importance. It is no longer believed that the reaction here marks, as it does in some other immunizing processes, a successful elaboration of protective substances. There is no single point about tuberculin treatment upon which there is the same unanimous accord as the baneful effects of general and severe reactions. The aggravated symptoms of the disease and the grave depression that follows often require many weeks before the patient's previous condition is re-established. The rôle played in tuberculin treatment by focal reactions is much more difficult to define. The marked increase in the number and extent of the râles that frequently occurs in pulmonary lesions has led to the belief that severe tuberculin reactions may cause a sudden extension of the disease. There is neither anatomical nor experimental evidence to support such a view; indeed what evidence there is points against the possibility of its occurrence. We know how insufficient our methods of examination are and how far short they fall of giving us a true picture of the extent of pulmonary lesions. Unquestionably the râles develop as an expression of the inflammatory reaction about pre-existing tubercles. It was these focal reactions that were looked upon by Koch as the essential feature of tuberculin treatment. His view was that tuberculin acts specifically not upon the tubercle bacillus but upon the tuberculous tissue, causing its death and disintegration when it is either absorbed or discharged from the body. In this way healing was thought to be brought about. However, the obstinate persistence of the newly formed râles following a severe focal reaction, usually associated as we have said with marked aggravation of the general symptoms, has led to the

belief that they are decidedly undesirable. Observation of external tuberculous lesions has taught us that even after doses that produce no apparent effect upon the general condition or temperature of a patient may cause appreciable change about the lesion. Such slight and frequently repeated inflammatory reactions may be of the highest importance in bringing about healing of the tuberculous focus. This reasonable assumption has been strengthened by the unquestioned improvement one will occasionally notice following a few slight general reactions. Such improvement is by no means constant and aware of the injury that we believe may follow severe reactions it would be foolhardy to attempt to produce them purposely. In our experience patients with pulmonary tuberculosis whose treatment has been frequently interrupted by recurring mild reactions have certainly not shown any more desirable improvement than those who have taken their course of tuberculin without a break.

In the treatment of pulmonary tuberculosis, then, and perhaps too in the treatment of other forms, our object should be to attain tuberculin tolerance and to reach this goal without the intervention of reactions. These principles are almost universally accepted, but different methods are employed in their application. Every one who uses tuberculin soon comes to adopt individual deviations from a given routine, but the methods may conveniently be divided into two groups between which there is every shade of gradation.

The first group is represented by Löwenstein,[6] Bandelier and Roepke,[7] Schlossmann and others. We refer particularly to Löwenstein's method because it is embodied in the directions for use accompanying Hoechst's tuberculin and is therefore widely circulated. The object of his plan is to reach high doses of tuberculin in the shortest possible time. Minor details of treatment are held subservient to this prime object. He begins by giving diagnostic doses of tuberculin to find to what amount the patient will give a general reaction. This initial dose having been determined, after a rest of from ten to fourteen days, treatment proper is begun by its repetition or even with a dose a little higher. From this point on the dose is progressively and rapidly raised. If reactions occur the dose is repeated if necessary three or four times and then again increased. Slight reactions are not held to be contraindications for enlarging the amounts. Above all, the dose must never be

decreased for fear of stimulating hypersensitiveness and making further advance impossible.

The second group is represented notably by Trudeau,[2] Sahli,[4] and Denys.[5] Theirs is a much milder procedure than the one advocated by Löwenstein.[6] While the aim is to arrive at as high a grade of tuberculin tolerance as posible, the reaching of large doses is not the ultimate object. Each patient is carried as high as his own individual tolerance will permit and is never forced onward through reactions. Treatment is begun with doses so small that no reaction will be produced and then cautiously raised, the slightest evidence of approaching sensitiveness being watched for. When these occur the amount of tuberculin is reduced or at least held at the same level until the indications have completely disappeared. The essential feature of the plan then is to avoid even the slightest reaction and instead of attempting to reach an absolute high dose of tuberculin to carry each patient to the measure of his individual tolerance.

Our work during the past three years has been entirely amongst ambulant patients and the experience we have gained makes us urge the adoption of the latter method. On at least two occasions, influenced by the apparent benefits of a more rapid ascension in dosage, we have become impatient and pushed the treatment more vigorously. The results were anything but advantageous and have forced on us the conviction that at least amongst ambulant patients the mildest method is the most desirable. Numerous reactions occurred and after some severe ones it was a long time before the patients completely regained their vigor. Besides this unfavorable result reactions, if at all severe, tend to break down confidence in the benefits of the treatment and cause many patients permanently to abandon it. The fear that small doses encourage the development of the hypersensitive state, a condition we shall speak of more in detail later, seems to us to be exaggerated. With a slow and gradual increase in amounts patients can as a rule without difficulty be brought to tolerate high doses of tuberculin. Tolerance too is frequently established without the slightest inconvenience to the patient and without a single break in the plan of ascension. A longer period is required, but the aim is accomplished just as surely and certainly more smoothly than with Löwenstein's method. It may be questioned whether the hypersensitive state develops less

frequently with rapid progression. We certainly feel that reactions and especially repeated reactions are more potent in raising sensitiveness than the repetition of small doses.

The avoidance of reactions, then, is the crux of successful tuberculin treatment. In spite of the greatest care mild reactions will occasionally occur but the signs of developing sensitiveness are so delicate and usually so evident that we can always avoid severe ones. A reaction to tuberculin consists of three essential features: a local reaction at the site of injection, general constitutional symptoms, and a focal reaction at the site of the lesion. In a severe and typical reaction all three are present but each may occur in the absence of the others.

The local reaction is the most sensitive and the most constant sign of approaching intolerance. It consists of redness, swelling, tenderness and pain at the site of injection. It comes on after from six to twelve hours and usually subsides in from two to four days, but may persist for a week and even longer. It varies from slight redness and just appreciable infiltration to painful nodules, often as large as walnuts with swelling of adjacent glands. The patient most commonly complains of a little tenderness when leaning against the point. It is, however, a matter of the greatest importance that a considerable local reaction may be present without the patient making any complaint of it. Before an injection is given the point of previous injection should be carefully examined. When a definite reaction has occurred the infiltration is apparent, but when the reaction has been slight it may be necessary to pick up corresponding areas of skin before it can be appreciated. We cannot urge too strongly the importance of these examinations. When the point of injection is carefully observed and used to guide us in the increase of dose, even mild general reactions become excessively rare.

Of the constitutional symptoms fever is the most important. Although by carefully watching for local reactions severe febrile rises seldom occur, unless the dose is too rapidly increased, it is important to pay strict attention to the least advance in the average daily maximum. A marked change in the mean daily variation is of less but still of some significance. If slight temperature rises, even of only a few fifths of a degree, are neglected, the following

injection may be followed by a severe general reaction. Patients with tuberculosis are so susceptible to temperature changes that it is often difficult to determine if a given rise is due to the tuberculin. Intercurrent infections should always be sought for. Tonsilitis, and during the winter epidemics of what are assumed to be influenza, have caused us some confusion. The latter infections, particularly, are often difficult in the beginning to distinguish from an aggravation of the pulmonary disease. The subsequent course of the attack, however, readily establishes the diagnosis. Denys is unwilling to attribute to tuberculin any febrile rise that comes on more than forty-eight hours after the injection. The rise of a febrile reaction to tuberculin is usually abrupt, even though the fall may be irregular and prolonged. A fever coming on with step-like progression so that the fastigium is not reached until the third or fourth day is unlikely to be due to tuberculin, although such reactions unquestionably occur. An abrupt febrile rise coming unexpectedly in the course of a perfectly smooth tuberculin treatment, when there has been no warning local reaction or constitutional symptoms, must exceedingly rarely be due to the injection.

Constitutional symptoms other than fever frequently occur as forerunners of approaching intolerance. They are never striking and must be carefully asked for to be elicited. A feeling of lassitude, slight general malaise, loss of the usual bodily vigor, depression, unaccountable restlessness and nervousness, digestive disturbances, and notably progressive loss of weight are the most important.

A focal reaction may be manifested by symptoms and by changes in the physical signs. Pain in the chest, dyspnœa and increased cough and expectoration are the characteristic symptoms. These need not be directly due to the tuberculin but their presence demands caution. Much less commonly there is an increase in the number and extent of the râles. However, marked changes in the physical signs we have observed only after severe general reactions.

Having in mind the importance of these general principles of tuberculin treatment it is apparent that no routine method can be put into practice. The sensitiveness of each patient must be carefully studied and made the guide for the size and increase of dose. In the beginning of our work we started all patients with

0.000,001 Gm., but as reactions not infrequently occur in the thousandth of a milligramme we later made the initial dose 0.000,000,1 Gm. and in many cases 0.000,000,01 Gm. In robust patients with inactive lesions the larger initial dose may be given but those showing any toxic symptoms, such as fever, impaired general nutrition, digestive disturbances, etc., should receive the smaller. One's first impression is that straw splitting about such insignificant doses is an academic refinement, but practice will soon teach a profound respect for the smallest amount of tuberculin. He who takes the lesson before experience enforces it is already advanced in the secret of successful treatment. In the beginning one will often be able to double the dose until some evidence of intolerance arises when progress must be slower. Although reactions, particularly local reactions, are not uncommon to such small doses, severe ones practically never occur. When 0.000,01 Gm. is reached, ascent must be slower. From here on the increase may be by decimals, going from one one-hundredth to two, three, four and five one-hundredths of a milligramme. Such progression, however, is extremely irregular, the second dose being double the first while the tenth is only a one-ninth increase. It will be found too that reactions occur most commonly after the first few tenths where the jumps are largest. To make the advance more uniform an extra dose may be inserted between the first and second and between the second and third and then go from five to seven and from seven to ten. Brown has devised logarithmic tables that allow of a perfectly uniform progression in dosage.

With this plan it is often possible to carry patients to large doses without a single interruption. When, however, the slightest sign of intolerance arises we must exercise great caution in the further treatment of the patient. When local reactions or even mild general reactions occur after small doses—after doses under a tenth of a milligramme—the same amount may be repeated. If this injection is followed by no further reaction the dose is again cautiously raised; if a reaction ensues milder than the previous, the same amount may be repeated until all evidence of intolerance disappears, and then be increased; if a more severe reaction follows subsequent injections of the same dose, treatment should be suspended for at least a week or ten days and then be taken up with a

smaller amount. When reactions follow larger doses several repetitions will often successfully carry the patient over the period of sensitiveness, but the attempt is not without risk and is not to be recommended as a general method of procedure. It is always safer after a short pause to begin again with a smaller dose and by a gradual increase the point of previous reaction will usually be passed without difficulty.

As we have previously said, more rapid methods of immunization are widely advocated and practiced with apparent success in many institutions. Still amongst ambulant patients particularly we would strongly advocate this perhaps over-cautious plan. In spite of Löwenstein's contention insurmountable hypersensitiveness seldom occurs. In treatment with tuberculin by any method it will happen occasionally that in spite of every effort it will temporarily be impossible to increase further a patient's tolerance This condition is spoken of as the hypersensitive state. A given dose may be approached over and over again and if finally successfully passed this is accomplished only after great perseverance. It is here that our patience is most severely tried. After several failures to establish tolerance by returning to smaller doses and again gradually ascending, it is advisable to select an amount of tuberculin just short of the one that causes a reaction and repeat this indefinitely,—ten, twelve, fifteen times. This will often bring the desired result. However, at times all our perseverance seems to but stimulate the patient's sensitiveness. Doses that before were readily tolerated now produce reactions and the patient may gradually lose weight and suffer a return of symptoms. Under these conditions the best plan is indefinitely to suspend tuberculin treatment. After a pause of some months it may be again resumed and often with good results. Intercurrent infections call not only for a suspension of treatment, but as they tend to heighten sensitiveness when again begun it should be taken up at a lower dose.

Even moderately severe febrile and general reactions will be exceptional if the method here outlined is carefully followed. In spite of utmost caution, however, they will occasionally occur. One must then wait at least ten days after all symptoms have subsided and re-establish treatment with a much smaller dose.

The best interval to allow between doses is a disputed question.

Wright, from studies of opsonic response, advocates that at least ten days should be allowed. It is reasonable to assume that a certain length of time is required by the body to elaborate antagonistic substances, but until these processes are more fully understood we cannot apply them dogmatically in treatment. The production of tuberculin immunity has been carried out upon strictly clinical principles and experience indicates that the injection of small doses twice weekly gives satisfactory results. This has been our practice except that when large amounts of tuberculin are given, doses of ten milligrammes and more, we prefer to make the injections only once a week.

There is no absolute length of time over which tuberculin treatment should extend and no absolute final dose. Having in mind the object we wish to attain it seems improbable that much can be accomplished in less than five or six months. A patient unable to devote this length of time to treatment had best be advised not to begin. When improvement is satisfactory tuberculin may be continued for a year or longer with advantage. The dose we reach must be determined entirely by the tolerance of the patient, although we have never gone beyond one gramme. It must be admitted that this dose is chosen arbitrarily, for some have given as much as twenty grammes. Having attained a gramme we have in some cases repeated this amount every two weeks for some months. Such a procedure is, however, not always practicable for at times sensitiveness returns and in our experience it is then best to discontinue treatment. Only a small number of our patients have reached this maximum dose and many were treated for over a year. The average maximum dose attained by our patients, excluding the far advanced cases, was a little less than one hundred milligrammes. *Incipient* cases and those that show the greatest improvement seem to tolerate the largest amounts. Several patients even after many months of treatment have never reached as high as 0.000,01 Gm. While improvement will occasionally occur in spite of such unusual sensitiveness we have become impressed with its ominous prognostic importance. While it is never advisable to carry the dose of tuberculin beyond the point where reactions threaten further advance, it is always desirable to reach large doses or at least the full measure of a patient's tolerance. In some instances unusual improvement

has been absent until large doses were attained when it was rapid and striking.

While many methods of preparing tuberculin dilutions have been advanced, the simple decimal system has seemed to us most satisfactory. We feel that it is desirable that the dilutions be prepared by the physician himself. The procedure is extremely simple, takes very little time, gives a more accurate idea of the actual tuberculin content of each dilution and saves considerable expense. Bottles, a one-tenth c.c., a ten c.c., and a one c.c. pipette are the only apparatus needed.

Our dilutions are made so that each bottle is one-tenth as strong as the preceding number. Number 1 is the pure tuberculin; number 2, 9 c.c. diluent and 1 c.c. tuberculin; number 3, 9 c.c. diluent and 1 c.c. of number 2; etc. The diluent is 0.8 per cent. salt solution with 0.25 per cent. carbolic acid. This scheme is simple and very convenient, as the number of the bottle represents the number of figures one must write to express the amount of tuberculin in each c.c. Thus bottle 5 contains 0.000,1 Gm. tuberculin to each c.c., bottle 7, 0.000,001 Gm. per c.c. etc. The dilutions are made in wide-mouthed, glass-stoppered bottles and are kept in a refrigerator when not in use. The salt solution must be carefully prepared with distilled water and pure sodium chloride. If any impurities are present they may cause endless annoyance by producing a flocculent precipitate which does not become evident until after twelve to twenty-four hours. If the pipettes, bottles and syringes are sterilized there is no danger of contamination. Fresh dilutions are prepared every two weeks. We have been unable to note any change in strength during this period.

Injections are made *subcutaneously* under the skin of the back in the region of the angle of the scapula, varying from right to left. The most delicate guide we have to tuberculin intolerance is the reaction at the site of the injection and it is important that this be where we may see and also feel it. For making the injections we have used syringes devised by the Randel-Faichney Company. These have a narrow bore of 1 c.c. capacity divided into hundredths. Of course one cannot measure accurately 0.01 c.c., but one can 0.05 c.c. Preparatory to injection the skin of the back is cleaned with alcohol. No dressing is applied. In making over 5000 injections we have never had the slightest infection.

One important caution should be given. Whenever changing from one brew of tuberculin to another, even if produced by the same laboratory, always diminish the dose. Tuberculin cannot be satisfactorily standardized and different products vary so much in strength that severe reactions may follow the repetition of the previous dose when a change is made. We have had one such unpleasant experience.

While patients are receiving tuberculin it is essential that accurate records be kept of their temperature and of their daily symptoms. In institutions this is readily carried out by the nursing staff, but ambulant patients may be taught to make satisfactorily their own observations. For convenience and permanent preservation special record books are desirable. An excellent one has been devised by Brown of Saranac Lake and is on sale by the *Journal of Outdoor Life*. We have modified his form to better suit conditions in the dispensary and give additional information about the diet and special features of the daily life. It is perhaps needless to add that during tuberculin treatment the usual hygienic and dietetic regulations should be as fully as possible enforced. These are the essential and fundamental principles in the management of tuberculous patients. Tuberculin can never replace them and can never be made an excuse for the least laxity in their application.

A wide difference of opinion exists as to the class of patients that may be benefited by tuberculin. We have tried to emphasize that tuberculin is no cure for tuberculosis and too much need not be expected from its use. What benefit it gives is brought about by an active response of the body cells and an individual already overwhelmed by the disease cannot produce such response. In our experience the general condition of the patient is a much more important factor in predicting the results of tuberculin treatment than the extent of the disease. In patients who are ill, with fever, rapid loss of weight, and great prostration, and in whom the disease is rapidly advancing, nothing can be accomplished with tuberculin. The patient must be offering some resistance to the infection before we can hope for benefit. It is always best when fever is present to attempt its reduction by rest in bed before beginning tuberculin. When a slight daily rise of temperature persists and other conditions are favorable tuberculin will at times inaugurate decided improvement. Such an occurrence, however, is exceptional and where rest

in bed fails to reduce fever tuberculin seldom does good. The most desirable field of tuberculin application is in incipient cases. Sahli even urges its prophylactic use among suspected individuals and those who have been intimately exposed to the disease. The results are most striking, however, in certain inactive moderately and far advanced cases.

Other than the general condition of the patient we know of no absolute contraindication to the use of tuberculin. A tendency to hæmoptysis is not increased and unless the hemorrhage be large need not deter its use. Pregnant women when cautiously treated do particularly well on tuberculin. Epileptic seizures are not more frequent or more severe. Compensated valvular disease of the heart is certainly not unfavorably influenced. We have never treated patients with nephritis, but this we would certainly consider a strong contraindication. We have never seen or heard of an ill effect due to the careful use of tuberculin upon the cautious plan outlined.

In conclusion it is well to emphasize again that tuberculin immunity is not tuberculosis immunity. Not only is it possible for the original lesion to spread while the patient's general condition improves under tuberculin treatment, but even when large doses are tolerated the disease may break out in other organs. One of our patients developed tuberculous laryngitis after doses of twenty and thirty milligrammes had been successfully attained. Another after having greatly improved under tuberculin administration suffered a severe relapse after reaching thirty milligrammes.

SPECIAL REFERENCES

[1] Koch: Deut. m. Woch., 1891, p. 1191; *Ibid*, 1897, No. 14.
[2] Trudeau: American J. M. Sc., 1906, vol. cxxxii, p. 175; Sixth International Tuberculosis Congress, Washington, 1908, vol. i, part 1, sec. 11, p. 796.
[3] Spengler: Deutsche med. Wchnschr., 1905, vol. xxxi, pp. 1228 and 1353.
[4] Sahli: Uber Tuberkulinbehandlung, Basel, 1907.
[5] Denys: Le bouillon filtré, Louvain, 1905.
[6] Löwenstein: Handbuch der Technik u. Methodik d. Immunitatforschung, Kraus u. Levaditi, vol. i. p. 821.
[7] Bandalier and Roepke: Lehrbuch der Specifischen Diagnostik u. Therapie der Tuberkulose, Wurzburg, 1908.
[8] Brown: Specific treatment of Tuberculosis, Edited by Klebs, D. Appleton & Co., 1909.
[9] Hamman and Wolman: Tuberculin Treatment among Dispensary Patients, Johns Hopkins Hospital Bulletin, August, 1909.

A GENERAL CONSIDERATION OF THE THERAPEUTIC USES OF DIET

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The assertion will be admitted that, as a rule, dietetic measures are employed rather carelessly as compared with the exactitude and consistency with which drugs are prescribed.

That a faulty diet will produce disease, and that abused digestive organs, in their rebellion at unfair treatment, can set in motion a train of morbid symptoms affecting nearly every function of the body is a recognized fact.

Acute manifestations of disease require medicinal remedies mainly, but there is present at all times the problem of the bodily metabolism. No matter what conditions arise, the human furnace must be stoked, and heat and energy furnished, or we perish. The vegetative functions of the body must be sustained, while Nature, our most valuable ally, is endeavoring to swing the jarring and discordant elements into that equilibrium called health.

As Sir James Crighton-Browne recently said, "One of the most important questions is 'what must I eat to be saved?' a question to which answer is made by a shouting multitude of enthusiasts, cranks and empirics, each with an infallible dietetic system of his own."

Now I do not propose to touch on any of these extraordinary systems, nor will the limitations of space permit me to enter the lists in the battle now being waged between the exponents of the high- and low-proteid ration. I simply desire to confine my remarks to a general consideration of diet as a therapeutic measure.

We may roughly divide the food of man into five classes,—protein, fat, the carbohydrates (starch, sugar, etc.), minerals, and indigestible portions, or refuse. Each one of these, even the waste, has its useful function, and when all are furnished in proper quantity and proportion to a normal digestive apparatus, there results a vigorous well-nourished body. There is, however, a considerable variation allowable both in amount and quality, and,

where the organs concerned are in good condition, any excess is either stored away, or carried off with the refuse; while any deficiency is met by a draft on the reserve already provided. In either event the nitrogen balance remains practically the same.

For these reasons the natural appetite in healthy people is a fairly reliable index to the bodily needs, and a rational craving should therefore be heeded. In this connection I might add the following from a recent address of Dr. H. W. Wiley: "I take it as a proposition difficult to disprove, and sustained by every principle of analogy and reasoning, that the ordinary normal diet of man, selected by the necessities of nutrition and by taste, is considered all in all the best."

When from direct or indirect causes, however, the digestive organs become incapable of performing their special duties, there ensues such a state of disorder that the appetite can no longer be relied upon, and the body either lacks the substances which should be made ready for use by the incapacitated organs, or is irritated by excessive undigested residue.

Under these conditions, no matter what medicinal or other measures may be employed, it is obvious that *intelligent* dietetic therapeutics are of the utmost importance.

The unsystematic method is to be deplored in which many otherwise careful practitioners set in motion a dietetic regimen, allowing too much latitude to the appetite, not noting if sufficient calories are being ingested; or, if some particular part of the alimentary tract is crippled, not seeing to it that the food element taxing that part is either lessened or omitted.

Suppose mastication is hurried and careless, the food lacking in salivation, poorly comminuted, is rushed down the œsophagus like coal down a chute. This calls for the first dietetic injunction.

Suppose after the food reaches the stomach discomfort ensues, either immediately, or in one or more hours. This discomfort certainly indicates a minus or plus condition of the stomach, shaded by many modifying influences. Should the motor power of the stomach be impaired; should an ulcer or inflammatory state of the mucosa be present; should the hydrochloric acid be in excess, deficient, or absent altogether; should the organic acids be too much in evidence, then any of these abnormal conditions may be aided, and often cured by suitable diet alone.

Suppose the liver fails in its sentry duty, or the pancreas is derelict, so that the small intestines, handicapped by the products of fermentation and putrefaction, cannot extract their toll, and, instead of normal metabolism, there sets in a state of catabolism, with its manifold symptoms. Here a cleansing of the bowels will serve like rubbing a faulty sum off a slate, but, unless a proper diet is furnished, the same disturbances will speedily reappear.

Many cases of constipation are directly traceable to a concentrated diet, which leaves so little residue, that there is nothing in the large intestine to stimulate peristalsis. The muscular fibres of the intestines, as much as the voluntary muscles of the body require regular work to maintain their tonicity, and, like the other muscles, soon become inefficient, when deprived of their necessary exercise.

The general status of the patient should have weight, for in the robust, or in those where there is a plentiful reserve of fat, certain offending elements may be eliminated from the dietary without fear of unduly lowering the vitality. Often in the obese, whose digestive organs for years have had to split up complicated menus, or in manual laborers, accustomed to consuming immense amounts of proteids, complete abstinence from all foods for three to five days, though allowing plenty of water, will frequently give almost magical results.

I well remember a very obese patient, seen with Dr. E. L. Kellogg, of New York City, who was covered with purpuric splotches, the urine loaded with indican, and who said he felt as if "the boiler was about to burst." This man was given a saline laxative, and ordered to eat nothing for seventy-two hours, but to drink plenty of water. At the end of that time he reported much improvement, and was allowed a diet of toast and milk for two days, after which he was given no food for another period of seventy-two hours. At the expiration of the second fast, his skin was clear, his urine normal, his step firm, and he said he felt better than for years. Though he complained of an empty sensation in his epigastrium, he said that he had suffered but little from actual hunger.

Not a few who read this article will recall former cases of chronic indigestion, who, after some continued illness, which forced

them to give their alimentary tracts a long rest, were agreeably surprised to find their indigestion entirely cured.

In most acute diseases, accompanied with fever, the waste products of the body are excreted in excess, while constructive metabolism is in abeyance. The digestive fluids are diminished in quantity and efficiency, the assimilative powers are below par, while the appetite is entirely lacking. In these conditions only enough calories should be furnished to stem the tide of retrograde metamorphosis; and, as all nourishment must assume the fluid form before it can be absorbed, liquid food is best suited, because it places a minimum of labor on the crippled digestive machinery. I might state as a general dietetic principle, that in acute febrile conditions solid food should be given sparingly, if at all.

In considering liquid food, let me sound a warning note against the various 'extractives' as dependable nourishment. I agree entirely with Dr. A. L. Benedict, of Buffalo, who says: "A meat broth prepared at a temperature above 160° F., the coagulation point of albumen, contains salts, extractives, which are mainly excrementitious, and a little gelatine, as well as some melted fat, although the last is often skimmed off to make the broth more pleasing and palatable. In so far as proteid is concerned, a meat tea made by boiling cannot be more nourishing than egg tea, that is to say the water in which eggs are poached, or in plain words, it contains no proteid nourishment at all, and is, barring certain qualitative and quantitative differences, of the same dietetic value as urine."

I have frequently seen patients fairly water-logged with soup, and who were really getting less than 100 calories daily. When such cases recover, it is brought about by the contributions of the reserve proteid and fat stored in the body, and not from any decided nutritive power of the soup.

The presence of anorexia will often present knotty problems to the student of dietetics, especially that form remaining after acute disturbances have been corrected. It is here that the personal equation must be noted, the likes and dislikes allowed for in the food provided, while in serving, no detail of the æsthetics should be omitted.

In anorexia nervosa the question of diet is not one of quality but of quantity, any concession to a capricious appetite being

allowable, provided enough is ingested. Occasionally, when the appetite has completely fled, the yielding to a craving for some bizarre article will favorably turn the scales, and pave the way for a normal hunger. Should food be refused until starvation is imminent, special methods of alimentation are indicated, the description of which would hardly come within the scope of this paper.

Those suffering from wasting affections, convalescents from acute infectious fevers, victims of long-continued body- or brain-fag—all that class of invalids, in which the oil runs low, and the vital spark shines but dimly, need a diet far in excess of the daily caloric requirements. Proteids and fats in such cases should predominate, and should be in a form easily assimilable. All the tissues, without exception, find constant use for fat-cells, utilizing them at every stage of their metamorphosis; and, if fat in sufficiency is provided, the digestive organs are not forced to ransack each nook and cranny of an already depleted body, like a scouting party of an army seeking to replenish the commissary department in a country impoverished by previous warfare.

Where the wasting process is active (pulmonary tuberculosis, for instance), supportive measures must be strenuously pushed up to the point of suralimentation. The doctrines of physiological economy may be held secondary to the necessity of aiding the body in its battle for strength to overcome the malign forces of disease, which are constantly sapping it.

The late General N. B. Forrest used often to say that the most successful commander was the one who got there first with the most men, and the physician, who, early in the disease, actively supports the body by a generous diet even up to the point of forced feeding, will much more often see his patient win the fight for health.

Among the autointoxications, especially the intestinal, some general suggestions may be profitably offered. As the animal proteids are more liable to putrefactive changes than are the vegetable, if the patient is changed for a time to an exclusive vegetable regimen, a marked improvement may be expected. An ideal diet for man combines both classes, but it must be admitted that the flesh proteids often have to be omitted in toxic conditions, before a foul intestinal tract can be corrected.

Artificially fermented milk, fills an important place in such conditions, and the fact that just now it is a fad, with all the attendant exploitations, should not detract from its field of usefulness.

The dietetic uses of alcohol, fraught as they are with social and economic complications, will not be specifically discussed, but I will go on record as saying that I do not consider alcohol a suitable food, and that in wasting diseases its benefits do not go beyond the stomachic effects produced by small doses.

My own results in the use of dietetic regulations have convinced me that there is a normal diet for an individual, and this should be studied as one studies a psychic phenomenon. Previous habits in regard to articles eaten, as well as the time of meals, should be conformed to as much as possible, for, unless the patient is the victim of a vicious circle, or is addicted to a very harmful mode of life, it is not well to institute changes too revolutionary in their character. No one article of food should be forbidden unless there is a good reason for it, and, generally speaking, a reason that can be explained to the one most concerned. An injunction to give up some cherished habit, or forego some highly prized delicacy, should be fortified by more than the plain *ipse dixit* of the physician.

The laity of this country are reading much along dietetic lines, and there are frequently found in the popular magazines splendid articles, couched in untechnical terms, containing information of practical value. In many of the schools for girls and young women there are departments of cookery, where the basic principles of dietetics are now taught along with the culinary art, and if the medical man of to-day and to-morrow does not apply himself to a scientific study of this important branch of supportive therapeutics, he need not be surprised to find himself being instructed, as it were, out of the mouths of babes and sucklings.

Furthermore, I do not believe the day is far distant when dietetics will constitute a separate and respectable branch of every medical curriculum, and each student will be made proficient in all that relates to the intelligent nourishment of the body in both health and disease.

HYPNOTISM AND PSYCHOTHERAPY

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Hypnotism has been practised for generations. The ancient Egyptians had a profound knowledge of the subject, and it was largely used by their priests to impress upon the people the belief that they were possessed of supernatural powers. It is believed by many that the so-called miracles of converting rods into snakes performed by the Egyptian priests was a result of their hypnotic powers. All the nations of the Far East have from time immemorial known of hypnotism, and have used it through their religious men to induce the people to believe that they were endowed with occult power.

Anthony Mesmer, in Vienna more than a century ago, introduced a method by which he claimed consciousness could be suspended in man and in the smaller animals, and what was known as the "mesmeric" sleep produced; under the influence of which the "medium's" thoughts, will, and actions could be modified.

James Braid, an English physician, in 1843, was the first to investigate the whole subject of hypnotism thoroughly and scientifically. Since that time hypnotism has been studied by many scientific men, and the matter has been placed on a more or less scientific basis.

Buckley sums up hynotism as follows: (1) The early ideas of hypnotism were based on an erroneous doctrine. (2) The phenomena are governed by natural law. (3) There is absolutely nothing mysterious except to the unintelligent. (4) It is a partial suspension of consciousness. (5) The condition seems to simulate the sleep of some persons, best illustrated in sleepwalking. (6) The co-operation of the subject is necessary for inducing hypnotism. (7) Ill effects have been known to follow the practice of hypnotism. (8) It should not be practised for amusement or gain. (9) The best way to govern the practice of hypnotism would be by law.

The condition induced by hypnotic influence is defined by Lee as an abnormal excitation of hysterical disturbances of consciousness, and even what may be called epileptiform conditions of forgetfulness, and in some cases double consciousness. The hypnotized individual receives impressions while in a state of sleep-hypnotic trance. It is an abnormal condition, and therefore a very dangerous condition, and should be used only by those who are specially qualified by long training to practise it.

Psychotherapy is the remedial influence springing from the application of hypnotism or mental influence for the relief and cure of ailments. The proper execution of this procedure demands a due appreciation of the exact nature of hypnotism, and through hypnotism, suggestion. At the best hypnotism can merely be allied to an induced sleep by the agency of suggestion. During the period of repose, as in sleep, judgment and inhibition being in abeyance, suggestions made to the subject are carried into effect by the receptive faculties still remaining intact.

It is absolutely essential in the practice of hypnotism to gain the confidence of the subject, and for this reason it is asserted authoritatively that intelligent subjects lend themselves more readily to hypnotic influence than ignorant individuals. Preliminary to inducing the hypnotic state it may be advisable that the prospective subject should witness such procedures, have impressed upon him the ease of their execution and the lack of after effects of any ill nature.

To bring the individual under the suggestive control he must be put into the hypnotic state. A good method is to secure a fixation of the eyes, that merges into drowsiness after a while bordering on sleep. At the moment when a vague expression manifests itself, gentle pressure is to be made on the lids and the forehead slightly crowded down upon the lids.

By gently stroking the extremities, the suggestion of a tired feeling is imparted to them. The suggestion as to the disappearance of any ailing ought to precede hypnosis.

The use of hypnotism in the treatment of disease was first initiated by French physicians, and they are still the leaders in this form of treatment. At Nancy there is a school founded by Liebeault, in which hypnotism is taught as a branch of the medical

curriculum. In 1878, Charcot, the French neurologist, began his studies of hypnosis in the treatment of hystero-epilepsy, at the Saltpétriére Hospital. His observations were confined to that class of subjects, and he taught that it was a morbid neurosis, almost always on a soil predisposed to hysteria.

According to Auguste Voisin of the Saltpétriére, no one believed it to be possible to hypnotize the insane until 1880, when he first succeeded in a case of acute mania. In 1882, Esdaile reported that he had hypnotized thirty-seven insane patients in the Calcutta Asylum. From 1880, Voisin devoted himself largely to the treatment of mental disorders by hypnotic suggestion. Repond, of the Cantonal Asylum of Maiseus, Fribourg, claimed to have obtained good results in insane patients who were not hysterical, a result not previously obtained. Buckhard, of the Prefargier Asylum, recorded successful cases of delusional insanity and acute puerperal mania treated by suggestion. Liebeault, of Nancy, reported good results from the treatment of suicidal monomania by similar means. Smith and Myers published in 1890, an account of the hypnotic treatment of twenty-one insane patients at the Bethlehem Hospital, London. Robertson, in 1893, recorded similarly good results at the Morningside Asylum, Edinburgh.

According to Robertson, the following is a summary of indications for the therapeutic employment of hypnotism:

1. As a direct therapeutic agent: (*a*) in insomnia it may succeed in intractable cases where narcotics have failed (as hypnosis is more closely allied to healthy than to drugged sleep, it must be of great service where the brain nutrition is already bad, and the effect of depressing drugs is undesirable); (*b*) when the brain is in a highly unstable condition it may be of direct therapeutic value as a sedative, in preventing an outburst of excitement from passing into mania; (*c*) in dispelling fleeting delusional states, and in the minor psychoses it may be of service.

2. For purposes of management: (*a*) in overcoming the morbid resistance of patients for their own benefit (for example, as regards the administration of food or medicine); (*b*) hypnotism may be used in cases of excitement and violence, instead of mechanical, chemical or physical restraint.

In the treatment of dipsomania, hypnotism has been employed

with some success. Ford claimed that he had obtained excellent and durable results in this disease. The recorded cases are chiefly French. Voison, Ladame, Tatzel, Hirt, Nielson, De Jong, Liebeault, Bernheim, Van Eeden, Van Reuterghem, Hamilton, Osgood, Lloyd, Tackey, Kingsbury, and Woods are among those who have reported favorable results from the treatment of dipsomania by hypnotism.

Morphinomania also is said to yield to the influence of hypnotism. Wetterstraud in 1896 stated that, out of 51 cases of this condition treated by him, 37 were cured. Fuld, Morot, and others have reported good results.

Of all the cases of successful treatment by hypnosis reported it is probable that half were of hysterical origin, but at best it is possible that hypnotism in these cases is but an adjunct to treatment.

Berillon states that for three years he watched the hypnotic treatment of *la grande hysterie* by Dumontpallier at the Pitie Hospital. In the majority of cases, not only did the convulsive attacks disappear, but the other symptoms also. Voison, Burot, Burckhardt, De Jong and many others have testified to the benefit derived in the treatment of hysteria by hypnotic influence.

The use of hypnotism is indicated in cases of obsession. Milnè-Bramwell, as well as numerous European medical men, have recorded good results with patients suffering from this form of mental trouble.

Vicious and degenerate children are often amenable to hypnotism, which not infrequently has a lasting effect. Lyon, after referring to the fact that overexertion, mental or physical, and professional emotions are sometimes the sole causes of dyspepsia, and almost inevitably aggravate or perpetuate it, states that in such cases psychotherapy is the most important weapon at our disposal. When the gastralgia is a psychoneurosis it is the only treatment needed, and in cases of actual gastritis, it acts on the organic lesion. One of the aims of psychotherapy should be to divert the patient's attention away from his stomach, while inspiring him in the desire to recover and the confidence in a cure.

Aggadzauiautz has succeeded in arresting skin diseases by hypnotic suggestions which restored this sensation to the normal. The patients who were subjected to this treatment, included both

hysterical individuals and persons without symptoms of hysteria. The cases selected were those in which definite causes, such as parasites, disturbances of digestion, etc., could be excluded, and no local or general treatment whatever was used as in each instance every measure tried previously had failed. Aggadzauiautz concludes that mental therapy is an important addition to the methods of treatment in skin diseases. Mental suggestion acting upon the circulation and increasing the appetite, strengthens the organism, and especialy the skin, and this aids in the treatment. When the sensibility of the skin is lowered locally, various skin eruptions are often seen, which run parallel to the degree of the disturbed sensibility. Mental suggestion acting upon the latter, indirectly assists in the cure of the skin disease.

As to whether hypnotism can cure insanity in its graver forms, is a question which has been by no means settled.

Bramwell says that in endeavoring to estimate the value of this form of treatment, it must not be forgotten that our knowledge practically dates from Voisin's first case in 1880, and that comparatively few attempts have been made to give the method a fair trial. Certain forms of insanity are undoubtedly curable and it seems reasonable to suppose that a method of treatment which can induce sleep without the aid of drugs, and remove excitement and other morbid mental conditions, may prove a valuable remedial agent. It is doubtful also if epilepsy is curable by hypnotism, although numerous statistics have been brought forward in support of this statement. Therapeutic results with regard to other diseases are fairly satisfactory. With regard to susceptibility to hypnotic influence, Bramwell says, "As the result of extensive statistics we are apparently justified in concluding that the majority of mankind can be hypnotized." In 8705 cases reported by fifteen observers in different countries, only 6 per cent. remain uninfluenced. As to the time required to place a subject in a condition of hypnosis the same authority says, "In a small proportion of cases, hypnotism is easily and rapidly induced, but as a rule, the length of treatment required bears a direct proportion to the duration of the illness and the mental instability of the patient."

Authorities see no danger in hypnotism itself, but only in the suggestions which may be comparable to the knife in unskilled hands. The hypnotizer as well as subject should always have a

third party present for legal reasons. Hypnotism is not as harmful as the use of morphine or even chloroform which also induce sleep.

For the relief of the pains of tabes, if only temporary, an easement of the mind is brought about which insures sleep, and inspires confidence in the wake of which follow good appetite and increased strength. Instances are cited of the capability to render labor painless. But above all the domain for hypnotism is the relief of minor ailments, aches, agonies and perturbations of the mind, not associated with any distinct organic condition. The tractable minds of children are particularly amenable to hypnotism and chorea is cited as being easily controlled by hypnotic suggestion.

The ease with which hypnotism is effected depends upon the skill of the hypnotizer, who must be endowed with endless patience and rich in resources to give varied form to his suggestions making for a cure.

Anæmics, subject to a host of minor ailments which accompany their disease, are well suited for cure by hypnotic suggestion. The pains of rheumatism and the depression of tuberculous patients are also open to the influence of hypnotic suggestion. This is taking advantage only of the psychical phenomena of these diseases; their organic origin remains untouched and therefore requires medicinal treatment.

Nervous headaches and severe neuralgias in due process of time yield to hypnotic suggestion.

This enumeration it may be said, is merely claimed, not proven, for the modus operandi depends upon the searching nature of the hypnotizer to fathom the temperament of the patient, deport himself with dignity and never betray by word or deed any doubt as to eventual restoration. In short, the height of self control must be practised by the hypnotizer. The most commonplace and concrete instance of hypnotism is the use of medical agents void of any specific effect towards the particular symptom complained of, yet which are effective in a surprising degree, in the removal of the disturbing phenomena.

These measures we are wont to call placebos and they are legitimately applied by practitioners daily. This is exemplified in the following: The administration of aromatic spirits of ammonia three times daily in a healed fracture of the arm to strengthen the limb, and to be taken for two weeks. The patient at the expiration

of this interval of time returns benefited and asks for more medicine. The thoughtful physician complies, although thoroughly mindful of the futility of the agent. The taking of medicine thrice a day for an avowed purpose operates so powerfully on the mind, that the slow workings of time do not annoy him. Instances might be multiplied of such suggestive therapy, which can if necessary be carried to the extent of actual hypnosis. Honestly practiced *by the physician* only, psychotherapy, and not the hypnotism of the wandering showman, constitutes a legitimate practice and a powerful weapon in the hands of the scrupulous medical adviser. Much, but not many things, can be cured by some form of psychotherapy.

In conclusion we would cite the powerful influence that sound has on the mind. The use of the flute by the snake charmer is an instance under consideration. From this primitive yet fundamental use of music it is but a step to the sounding of a gong for a long period to induce hypnosis. This was practiced by Charcot at the Salpétriére. It is also an extensive practice in insane asylums to play music to the inmates. The martial music of the most primitive tribes and the sublime funeral dirge, alike bear testimony to the powerful operation of music on the mind in health and perhaps disease.

Angell well sums up the pros and cons of hypnotism as follows: Its greatest service will be found in dealing with morbid psychological conditions, such as fixed ideas, morbid habits, sexual perversion, etc.

Finally, public seances by professional hypnotists should be forbidden by law. Hypnosis should be employed only by physicians or psychologists familiar with the physiology and pathology of the mind. It should never be induced without the consent of the subject or his legal guardian. It should not be employed except in the presence of a third person. Suggestions which are contrary to the moral nature should never be employed. After the employment of hypnotism the patient should always be awakened and left in a calm mood. Regarding its medico-legal relations, it is pretty well established that in no case can a suggestion contrary to the moral nature of the subject succeed, the antagonistic instinct serving in turn as an overpowering negative suggestion.

Medicine

THE DIAGNOSIS AND TREATMENT OF PERNICIOUS ANÆMIA

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In entering upon a discussion of pernicious anæmia, one must recognize primarily that it is a condition rather than a disease, due to some obscure toxic influence and representing a greater or lesser degree of injury to the blood-making tissues, especially those concerned in the formation of red cells.

It seems proper to preface the consideration of the diagnosis and treatment of pernicious anæmia by a brief statement as to the present conception of the nature of the process.

While the ordinary classification into symptomatic or secondary, and pernicious anæmia prevails, essentially they are similar conditions differing largely in degree only; both anæmic states are secondary and evidently of toxic origin, the injurious influence in the former manifesting itself mainly as a hæmolysis but in the latter the toxic irritation is more prolonged and as the bone-marrow and other blood-making tissues become affected, a condition of disturbed hæmogenesis results. Pernicious anæmia as a disease condition represents an active hæmolysis as well as a disturbance in the genesis of the red cells.

Addison in his first description of pernicious anæmia stated that it occurred without discoverable cause, and even though more than half a century has passed by since then, the exact etiology is still a problem. From our present knowledge of its pathogenesis we recognize that the etiologic agent is essentially a severe poison that has a selective degenerative action not only on the blood-making structures, but also on the spinal cord, heart muscle, liver, and kidneys.

Pernicious anæmia is rarely found below the thirty-fifth year of life, and then affects mostly men. The large number of cases coming under observation at the Iowa University Hospital may suggest its greater frequency in rural or farming communities; but as Cabot correctly concludes from the study of statistics comprising 1200 cases, it is equally prevalent in cities, and more careful study tends to show it is commonest wherever most carefully sought.

Pregnancy, nervous strain, syphilis, chronic diarrhœa and other diseases are occasionally associated with it, but there is little evidence of any distinct connection. The enteric origin is supported by the frequently preceding diarrhœa, or other form of chronic gastro-intestinal disturbances; Herter attributes the pernicious anæmia to abnormal fermentation in the intestinal tract in which the anaerobic bacteria play a prominent rôle, but in spite of this, cases are seen in which there is no preceding diarrhœa or other intestinal disturbances, and again diarrhœa occurs often as a symptom, so that it is doubtful whether it is not more of a result rather than the cause.

Continued hemorrhage is a prominent factor in producing an anæmia, yet the type of anæmia resulting bears no great resemblance either in clinical picture or its course to pernicious anæmia.

To a certain extent pernicious anæmia lends itself to experimental demonstration. The work of Bunting, which has been corroborated here in connection with the working out of another problem, has demonstrated that a gradual long standing hæmolysis can be produced by the injection of small doses of ricin and saponin, accompanied by an erythroblastic metamorphosis in the bone-marrow similar to pernicious anæmia, and presenting a blood-picture like that occurring in man. The monograph of Schauman is a distinct contribution to our knowledge of the subject, demonstrating that a disease identical in all respects with pernicious anæmia can be produced by the presence of the fish tapeworm (Dibothriocephalus latus) in the intestine, and especially by the disintegrated particles of the worm.

A study of the pathology accompanying pernicious anæmia has been a great help in formulating the present conception of the nature of the process concerned. Regarding it as a toxæmia, the manifest change in the liver, kidney and heart muscle consists of

a fatty degeneration in the parenchymatous structure; in the spinal cord the degenerative change affects principally the posterior and lateral tracts, although occasionally a more diffuse myelitic degeneration takes place. The most characteristic pathological change occurs in the bone-marrow and upon this, the somewhat pathognomonic blood-picture of pernicious anæmia depends. By reason of a variation in the character of the changes produced in the bone-marrow, different types of pernicious anæmia result, and permit a classification as follows:

1. Hyperplastic or metaplastic pernicious anæmia: (*a*) erythroblastic—ordinary type; (*b*) leukoblastic—leukanæmia.
2. Aplastic pernicious anæmia.
3. Myelophthisic anæmia.

The bone-marrow being the main source of the red blood-cells, and chiefly concerned in pernicious anæmia, when subjected to injury responds by reason of its natural protective reaction, in the formation of immature cells or hyperblastic changes leading to the escape into the circulation of abnormal or poorly developed cells. Seen in the gross, the marrow is usually bright red, but microscopically it will be seen, that instead of the ordinary sized red cells with or without nuclei, a great number of larger cells (megalocytes), many of them being nucleated (megaloblasts) are noted throughout the marrow; from which it is assumed that the megaloblastic metamorphosis is the result of an inapt or unsatisfactory attempt at regeneration to offset the hæmolysis, or losses produced by the destruction of the red cells in the peripheral circulation; this call, chemotactic in nature, supposedly summons the marrow into increased and distorted activity, resulting in a hyperplasia, or still more a metaplasia, in which the place of the erythrogenetic centres is taken by cells of the fetal type (fetal reversion). Such is the change peculiar to the ordinary type of metaplastic pernicious anæmia.

That the above mentioned metaplasia of the bone-marrow does not always confine itself to the erythroblastic centres, is evident from the occasional observance of a case of pernicious anæmia suggesting a hyperplasia of the leukoblastic portions of the marrow, so that the crowding out of the fat is brought about quite as much by an excess of leucocytes as by the megaloblastic metamorphosis. Some of these newly formed leucocytes find their way into the

blood stream, presenting a somewhat puzzling clinical blood-picture, in which the features of pernicious anæmia and lymphocytic leukæmia are combined, giving occasion for the use of the name "leukanæmia."

In the aplastic type of pernicious anæmia the bone-marrow is a yellow color, and a fatty marrow is formed from end to end in the long bones, due to an atrophy of the erythroblastic tissue instead of a hyperplasia; apparently there is no reactive response on the part of the marrow to the injurious influence, or it is unable to make up the losses sustained by hæmolysis, and an atrophy results, which gives rise to a blood-picture presenting a decrease in the granular leucocytes, and a total absence of immature red cells, suggesting an unusual warped activity of the bone-marrow.

Myelophthisic anæmia is a rare condition in which there is an involvement of the bone-marrow by leukæmic nodules or some neoplastic growth, causing the erythroblastic tissue to be starved out or pushed to the bony wall enclosing it by the overgrowth of other cells, neoplastic or leukæmic. Because of the practical obliteration of the marrow, a profound or pernicious type of anæmia must necessarily result.

Diagnosis.—The diagnosis of pernicious anæmia is largely dependent on the recognition of those manifestations which must necessarily result from the changes incident to the pathogenesis of the diseased condition under discussion.

Its insidious onset, occurring mostly in elderly people, with the complaint of long standing and gradually increasing weakness, dyspnœa, marked pallor of the skin, and accompanying blood findings of an intense anæmia, constitutes the characteristic clinical picture of pernicious anæmia.

While the blood-picture may not be distinctly pathognomonic at all times, it nevertheless confirms the diagnosis, especially by finding the following:

1. A reduction of the number of red corpuscles to a point usually below two million per cubic millimetre.
2. A high color index.
3. A normal or sub-normal leucocyte count.
4. In the stained slide preparations, a predominance of abnormally large, greatly deformed, more or less abnormally stained red cells, some of which contain nuclei.

Certain characteristics noted in the stained specimen are typical of pernicious anæmia and these are:

a. Excess of over-sized red cells, which is really one of the most constant and characteristic blood changes in pernicious anæmia.

b. Abnormal staining reaction, a feature that is more frequently seen in this condition than in any other disease with the possible exception of lead poisoning.

c. Nucleated red corpuscles especially megaloblasts; while formerly the presence of megaloblasts was considered necessary for a positive diagnosis, it is now recognized that megaloblasts may be absent in otherwise typical cases of pernicious anæmia.

A characteristic feature in diagnosis, and of considerable prognostic significance, is the appearance of "showers" of nucleated red cells, also referred to as "blood crises," which first of all suggest a very profound type of anæmia, and again seem to appear as forerunners or warnings of the onset of a remission of improvement and again of a fatal termination; they are quite commonly observed in the experimentally produced pernicious anæmia.

In addition, the changes in the leucocytes become diagnostic criteria to a certain extent; a leucopenia is a common characteristic, counts as low as 400 white cells per cubic millimetre having been noted; with this leucopenia there is a striking reduction in the proportion of polymorphonuclear leucocytes, and corresponding increase in the mononuclear or lymphocytic type of cell, forming a state referred to as "leucopenie lymphocytose." This reduction in the protective polymorphonuclear leucocytes is only consistent with the hæmogenetic disturbance peculiar to pernicious anæmia, and in a prognostic way the "leucopenie lymphocytose" is significant of a grave form of the disorder. In studying the remissions of improvement peculiar to this diseased condition, it is noted that the leucocyte formula becomes modified as the general blood state changes for the better.

The diagnostic blood changes just referred to are peculiar to the metaplastic type of pernicious anæmia, and a differentiation into the other types is guided largely by the blood examination.

Aplastic pernicious anæmia is that form in which no doubt the same unknown hæmolytic agent is at work, but in which the marrow responds very faintly or not at all, and an aplasia or

atrophy of erythroblastic tissue is taking place instead of a hyperplasia.

This is a disease type more of young persons, women being affected oftener than men, running a rapid and progressive course, without any remissions of improvement, death usually occurring in the course of a few months. The blood picture shows a variation in that the (a) color index is generally low, (b) a leucopenia exists with the granular type of cells markedly diminished, while the lymphocytes are proportionately increased so that a lymphocytosis of 70 to 90 per cent. frequently prevails, (c) nucleated red cells are usually absent, and (d) poikilocytosis, anisocytosis, polychromatophilia is rarely seen.

As an illustration of this type of pernicious anæmia a history of a typical case is submitted:

Case I.—*Clinical diagnosis: pernicious anæmia, aplastic type, duration of illness one year, attending paraplegia.*

Mr. L. H., admitted to University Hospital May 18, 1907, being referred by Dr. Scripture of Cresco, Iowa. Complaining of weakness below the waist, inability to walk, and considerable trouble with the bladder and bowels. Patient is 30 years old, single, unmarried, does not use alcohol and denies venereal infection; family history negative. Former health good. Present illness began one year ago in March, 1906, by noticing a tingling and numbness in both feet as if they were asleep. This sensation has continued ever since, but has never been painful or changed into other forms of sensory disturbance. Worked well until August, 1906, and kept on doing light work until January, 1907, all the time getting weaker. Has not walked now for nine weeks, because of increasing weakness. Began to notice inability to move legs in bed four weeks ago, and now cannot raise them off the bed. Patient claims that the arms and hands are of late rather weaker than formerly and cannot feed himself very well, this weakness of the hands being of three weeks' duration. Appetite is fair, with no history of vomiting, diarrhœa or hemorrhage; bowels are rather constipated. Recently has had occasional trouble in starting urine or of controlling same if bladder is very full. During the past year has lost about twenty pounds in weight, formerly weighing 130 pounds.

The *present condition* reveals a young man of slender build of yellowish-white appearance; speech and mental state normal. Pupils react to light and accommodation; scleræ have an icteric hue. Tongue is very pale, moist, no tremor. Pulse rate is 100, regular, wave small, vessel wall soft. Chest slender, expansion fair; Litten phenomenon present on both sides. Lungs are normal. Heart is not enlarged, a systolic murmur being heard over the entire precordium, possibly best at the base, but is not transmitted. Abdomen is slightly distended and generally tender; liver and spleen are not palpable.

Extremities.—Arms are slender, of fair gross strength, with no tremor; biceps jerk normal, some paretic ataxia. The legs show a distinct œdema up to the knees; is practically unable to move any part of the legs at the toes, ankles, knees or hips. Attempts of movement at times cause a slight twitching of the united group of muscles. The knee-jerk is absent on both sides; ankle clonus is absent. Babinski reflex is present on the right side, no plantar reaction on left side. Moderate degree of ataxia. Tactile sensation disturbed; pain sense fair; thermal sensation indefinite. Urine examination reveals a moderate albuminuria.

The *blood findings* were as follows: hæmoglobin 30 per cent.; red cells, 1,040,000; leucocytes, 1800; differential count of 200 cells; polymorphonuclear 55 per cent., large mononuclear 22 per cent., lymphocytes 30 per cent.; eosinophiles 2.6 per cent. The stained spreads reveal a moderate poikilocytosis, microcytosis and macrocytosis, but no nucleation, stippling or polychromasia.

The patient became progressively worse, developed extensive bedsores, had an irregular daily fever, was unable to bear arsenic, even in small doses; had rectal and vesical incontinence and died May 29, 1907, twelve days after admission to the hospital.

An autopsy performed eight hours after death revealed but very little macroscopic change about the internal organs; microscopic sections of the heart muscle, liver and renal cortex showing a moderate degree of parenchymatous and fatty degeneration. Longitudinal sections of the tibia, femur and humerus revealed a yellow-colored marrow, which when examined microscopically showed a complete fatty metamorphosis of the erythroblastic marrow.

The brain and spinal cord revealed no macroscopic change, but microscopic sections stained by the Weigert-Pal method show a marked diffuse degenerative myelitic change in the anterior, lateral, and posterior columns of the spinal cord, the extensive degenerative process being further manifest by the involvement of the ascending cerebellar tracts in sections of the medulla. The accompanying plates of sections from the lower cervical cord and the medulla illustrate this very well.

This case is interesting in that it illustrates the type of aplastic pernicious anæmia, and also the unusual effect of the causative toxic agent on the spinal cord (Figs. 1 and 2).

The term leukanæmia is perhaps not well applied, presuming to indicate a condition intermediate between leukæmia and pernicious anæmia, with the clinical picture and blood analysis being usually more suggestive of the latter condition. As it is a comparatively rare affection, it may be of interest to report the following illustrative case:

Case II.—*Clinical diagnosis: pernicious anæmia; duration of illness one year; blood findings of aplastic type; leukanæmia; enlarged liver, spleen and cervical lymph-glands.*

Mr. B. S., admitted to University Hospital October 20, 1908, complaining of general weakness and shortness of breath; patient is a farmer, 68 years of age, married, four children alive and well. General family history is negative. Denies venereal infection. While in the service during the Civil War had frequent diarrhœa, and several attacks of malaria during which he was greatly jaundiced. Since leaving the army service has had several attacks of diarrhœa every year. For several years has noticed small lumps appearing on both sides of the neck which would vary in size from time to time. The abdomen has gradually enlarged during the past month. The present illness began about a year ago with a feeling of weakness and shortness of breath after any exertion. This has gradually increased and during the past three months has been bedridden a large share of the time; the kernels in the neck have become very much larger, some reaching the size of a walnut. Of late his pallor has become very noticeable, and he is now so weak that a walk across the room produces great exhaustion.

Fig 1.

Section of spinal cord, lower cervical region, from case of aplastic pernicious anæmia, showing diffuse myelitic degeneration.

Fig. 2.

Section of medulla from case of aplastic pernicious anæmia, showing especially the degeneration of the direct cerebellar tracts.

The present condition reveals an anæmic, emaciated and cachectic appearing man, the skin being of a lemon tint, sclerea faintly icteric, mucosæ and tongue very pale, mental state clear. On each side of the neck are six to eight enlarged cervical lymph-nodes, varying from hazelnut to walnut size, freely movable, non-adherent to each other and not painful. Several enlarged glands are palpable in both inguinal regions.

The chest is poorly nourished, symmetrical, of equal though limited expansion. A few dry râles are heard over the lungs posteriorly in the lower portion. A systolic murmur is noted over the entire præcordium, being best heard over the pulmonary valve area. A moderate degree of arteriosclerosis exists. The abdomen is generally distended, the wall is soft, in which several irregular nodules are palpable; there is no demonstrable ascites; the liver and spleen are readily palpable and distinctly enlarged. In the umbilical region an irregular, movable and non-painful mass about 8 cm. in diameter is palpable, that moves indistinctly with respiration; this enlargement suggests a thickened omentum. The extremities are slender, of soft musculature, fair gross strength with slight malleolar œdema. Station and gait normal, no tremor or ataxia. Knee-jerks are sluggish. No sensory disturbance. Urine findings are negative.

Blood, October 20, 1908: hæmoglobin (Dare 12 per cent., Tallquist 15 per cent.); red cells 410,000, leucocytes 5200; differential leucocyte count (200 cells) polymorphonuclear .7 per cent.; lymphocytes 78 per cent.; large mononuclear 21.5 per cent. October 21, 1908: hæmoglobin (Dare 12 per cent.), red cells 850,000; leucocyte 8400; differential count (1000 cells); polymorphonuclear .5 per cent.; lymphocytes 99.1 per cent.; large mononuclear 2 per cent. The red cells reveal a moderate poikilocytosis and anisocytosis, but no nucleation, stippling or polychromasia.

The patient left the hospital after a short stay and passed from observation, his death occurring about sixty days later at Livermore, Iowa. An autopsy unfortunately was not obtained.

The last two mentioned examples are essentially cases of pernicious anæmia, and aside from the varied blood-findings and clinical evidence of lymphoid hyperplasia, they differ from the ordinary type in degree only.

There are a series of symptoms in pernicious anæmia, like muscular weakness, dyspnœa, palpitation, headache, vertigo, tinnitus, anorexia, and œdema, which are peculiar to all severe anæmic conditions, and thus have no special diagnostic significance.

Gastro-intestinal disturbances, especially achlorhydria and diarrhœa, are more or less characteristic of pernicious anæmia; the absence of free hydrochloric acid in the gastric juice being almost a constant phenomenon; wherever diarrhœa is prominent, fever is also a special symptom attributable probably to absorption consequent to the abnormal intestinal fermentation.

Of special diagnostic significance are the symptoms referable to changes in the spinal cord, and there is no doubt if carefully sought for, the number of cases with spinal symptoms would be much greater.

Three groups of cases are observed:

1. Those in which the symptoms of anæmia precede and overshadow those referable to the spinal cord.
2. Those in which the spinal symptoms appear earlier, or give rise to more discomfort than those produced by the anæmia.
3. Those in which symptoms of this type are trifling or absent despite the presence, post mortem, of well marked lesions in the spinal cord.

There is no doubt that at times the spinal symptoms overshadow all others and it is not uncommon to see cases which have been diagnosed first, and not very improperly, as tabes dorsalis or as lateral sclerosis, and only by the development and recognition of the characteristic anæmia can this mistake be avoided. Again undoubtedly many cases are first brought to the attention of the physician by reason of the nervous symptoms before the anæmic condition is very manifest.

The posterior columns being principally involved, disturbances in sensation, like numbness, tingling, hyperæsthesia, paræsthesia and palasthesia in the feet, legs, and hands are common. These disturbances may in part be attributed to the general anæmia, but as a rule they suggest beginning changes in the spinal cord. More marked changes in the posterior columns are manifested by diminished knee-jerks and ataxic phenomena; as the lateral tracts become involved, a Babinski reflex, ankle clonus, and more or less

paralysis becomes manifest. That the degenerative process may be very extensive is evidenced by the illustrations accompanying Case I.

Aside from the sensory phenomena of hyperæsthesia and paræsthesia, there is a sensory disturbance to which special attention should be directed, and that is the phenomenon of palæsthesia or disturbance in the vibratory sense as elicited by means of the tuning fork. It has been the writer's experience to observe this quite frequently as the most prominent sensory disturbance and during a remission when other sensory disturbances would become less apparent, this palæsthesia underwent no change. Whether this is suggestive of a selective action of the toxin on special sensory paths, one cannot say, but yet is nevertheless an interesting observation.

Special types of posterolateral spinal sclerosis with profound anæmia are observed; in the so-called Putnam-Dana type severe anæmia, diarrhœa, delirium and symptoms of posterolateral sclerosis constitutes the symptom-complex.

By reason of the association of spinal cord changes and severe anæmias, I wish to submit a report of the following unusual case:

Case III.—*Clinical diagnosis: Putnam-Dana type of posterolateral spinal sclerosis; duration of illness one year; apparent severe anæmia with negative blood findings; diarrhœa; delirium.*

Mr. J. R. of Cedar Falls, Iowa, referred by Dr. Hansen, was admitted to the University Hospital January 23, 1909, complaining of inability to walk and general weakness. The patient is a farmer, 54 years of age, married, having four children alive and well. Does not use alcohol. Denies venereal infection. During the last six years he has had frequent attacks of diarrhœa. About a year ago began to lose in weight, appetite became impaired and periods of diarrhœa were more frequent with alternating constipation. During the past six months has been more or less bedridden, because of increasing physical weakness, so that now a walk across the room causes great exhaustion. Has been very much depressed in spirits and actions at times have indicated mental disturbance.

Present Condition.—Anæmic appearing, poorly nourished man of impaired mental state. Memory is poor, greatly depressed in

spirits and is troubled by a morbid fear of impending danger. Skin and mucus membranes are very pale. Eyes are normal; tongue is coated by a grayish fur. Teeth and gums are in poor condition. Temperature is 98° F., respiration rate 22; pulse rate 84, regular, vessel wall soft. No enlarged lymph-nodes. Chest emaciated, expansion fair. Lungs and heart normal. Abdomen retracted; slight tenderness over descending colon and sigmoid flexure region; liver and spleen are not palpable.

Extremities.—Arms are slender, gross muscular strength diminished; biceps and triceps jerk normal. Legs are very slender, the musculature being greatly diminished in size; gross strength weak. No ataxia. Knee-jerks sluggish; ankle-clonus and Babinski reflex present on each side. Station and gait uncertain, due to muscular weakness. Tactile sensation impaired; pain sense modified. Vibratory sensation absent. Urinary findings negative.

Blood.—Hæmoglobin 90 per cent.; red cells, 4,390,000; leucocytes 8,400; differential count 100 cells; polymorphonuclear 68 per cent.; large mononuclear 10 per cent; lymphocytes 19 per cent.; eosinophiles 3 per cent. Stained specimens reveal a moderate poikilocytosis, but no other special change in the red cells.

The patient remained in the hospital until his death on March 3, 1909, his condition continually growing worse, the diarrhœa became very persistent, finally leading to incontinence; the emaciation was extreme; delirium ensued, at times being maniacal in character. Bedsores developed and the focal signs of posterolateral spinal sclerosis became more marked.

Blood examinations were made at frequent intervals, and although the appearance suggested a profound anæmia, the red cell count was never found to be below 4,000,000 per cubic millimetre, and evidences of hæmolysis or hæmogenesis were not manifest.

At the autopsy anæmic and atrophic changes were noted in the heart muscle, liver, spleen and kidneys; the principal anatomical change was the evidence of a chronic colitis in the form of a general fibrous thickening involving principally the sub-mucosa of the descending colon, with numerous constrictions of the lumen. The brain and spinal cord showed no gross changes, but microscopical

sections of the spinal cord in the lower dorsal and cervical regions revealed small areas of fatty degeneration quite generally distributed throughout the posterior and lateral columns. The examination of the bone-marrow was limited to that found in the ribs, and this did not reveal any marked abnormal change. The diagnosis of Putnam-Dana type of posterolateral sclerosis was confirmed by the autopsy findings, the causative toxæmia being evidently enterogenic in origin, induced by the chronic colitis. The profound anæmia, though visibly present, which with the diarrhœa and mental disturbances usually constitutes the symptom-complex, was not supported by the hæmatological picture, yet I firmly believe this was one of those instances of anæmia where the entire volume of blood is reduced, without marked change being manifest in the proportionate distribution of its constituent elements.

Remissions.—Intermediate between the diagnosis and treatment of pernicious anæmia, and having a direct bearing on both, is the subject of remissions which forms one of the interesting chapters of this disease.

A remission signifies but an abatement of symptoms, and so rarely does a complete restitution take place that it is to be regarded as a temporary period of improvement and not a cure. The earlier writers who coined the term progressive pernicious anæmia, evidently did not give due cognizance to the phenomena of remissions, because a disease process that is subject to remissions of improvement, or periods of latency, can hardly be regarded as distinctly pernicious; the term progressive is applicable in that it is not possible to bring the process to a standstill or promote a cure.

Out of a series of cases observed during the past three years, I beg to illustrate the phenomenon by the two following charts (Case IV, and Case V):

A certain parallelism is maintained between the changes in clinical signs and blood findings, the blood condition being usually a fair index of the degree of improvement in the patient and also of the reactive changes in the hematopoietic organs, especially the bone-marrow.

Barjon and Cade and other French writers lay much stress on the value of the syndrome "leucopenie-lymphocytose" in diagnosis and prognosis. The disappearance of the leucopenia and

modification of the leucocyte formula suggests improvement and lessens the gravity of the prognosis; quantitative and qualitative changes in the erythrocytes being equally indicative of improvement.

Although the blood undergoes a marked improvement in the remission period, it still preserves certain abnormal features indicating that a disturbance in hæmogenetic function continues to exist. In this respect the experimental pernicious anæmias produced in rabbits differ from the disease occurring in the human person, it being found in several instances not only does an animal become tolerant to the toxin, especially with saponin in small doses, but if the toxic agent is discontinued the blood gradually returns

Fig. 3.

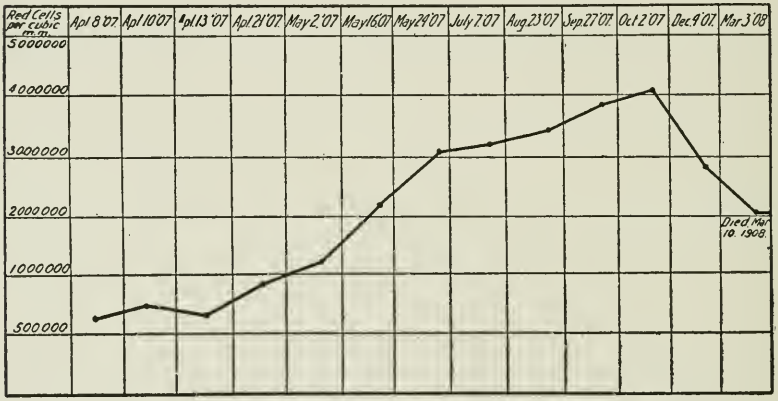

Chart of Case IV, showing clinical diagnosis: pernicious anæmia; blood findings metaplastic type; paræsthesia, hyperæsthesia, diminished knee-jerks; duration of illness one year; age of patient 53 years. Treatment: rest, Fowler's solution in small doses; no colonic irrigation.

to the normal and a complete restitution takes place, all of which is in keeping with the statement of Bloch, that certain predisposing conditions exist in the hæmatopoietic structures of the human species, which favor the production and persistence of pernicious anæmia in man.

It becomes an important matter to diagnose a remission and to recognize pernicious anæmia during the remission period. As the blood picture gradually becomes like the normal, and muscular weakness, with the perverted sensory phenomena disappears, it becomes very difficult to recognize the underlying condition. While there is a marked improvement generally, rarely, if ever, does a complete restitution take place. Even in observing the appearance

of the patient, though the pallor is less marked and the mucosæ have more color, there is still a dark anæmic-like hue about the skin that is distinctive and which will be found to be a common experience if carefully noted.

In all the cases reported referring to the remission phenomenon, there is no instance recorded where the free hydrochloric acid returned to the normal, indicating that a complete restitution to the normal in that respect does not take place. Unless one is conversant with the previous history of the patient it is readily to be seen how the pernicious anæmia condition could be overlooked.

FIG. 4.

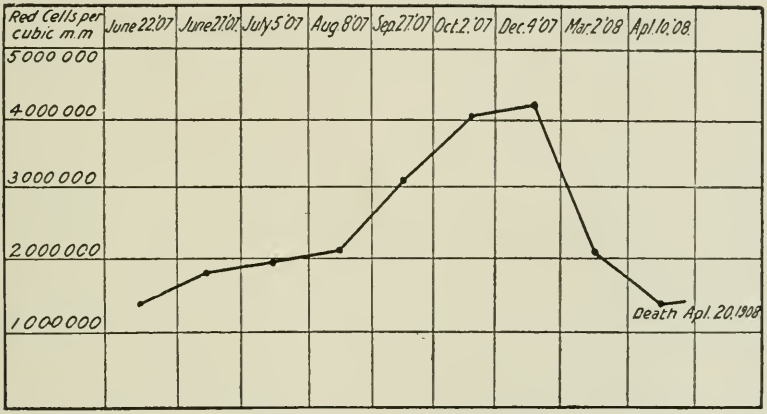

Chart of Case V, showing clinical diagnosis: pernicious anæmia—metaplastic type; duration of illness, one year and a half; enlarged spleen; farmer, age 45 yrs. Treatment: moderate rest, Fowler's solution of arsenic, no colonic irrigation.

I know of a recent instance where a very prominent surgeon in operating for gall-stones, entirely disregarded the diagnosis of pernicious anæmia and the careful blood examination made by a very competent internest six months previously, because, as he said, the patient looked too well, and furthermore he would not now be living if such were the case.

The average duration of remissions of improvement is three months, although instances are recorded where patients have remained comparatively well as long as three years.

Treatment.—While the present lack of unity prevails in regard to the cause of pernicious anæmia, the various plans of treatment will be based largely on our conception of the nature of the disease process.

It is but natural that the first rule should be to place the patient at rest because of the low blood count, physical weakness and general necessity for restricting all exertions. A great many patients improve if placed in bed, but on the other hand, a majority are in a sense already at rest, and the "rest cure" is therefore not applicable. There is no doubt that good hygiene, nourishing food and living out of doors tends to prolong life. In making some studies on metabolism in this class of patients, we felt for a long time that the keeping of patients on a purin-free diet relieved much of the intestinal fermentation, and it is likely that the avoidance of nitrogenous food tends to diminish the intestinal bacteria, but we have become convinced that no special improvement in the general condition can be attributed to this feature, and there is no evidence that any special diet has any influence on the course of the disease.

On account of the researches of Herter, attributing to anaerobic bacteria a prominent rôle in the intestinal fermentation, which is regarded as the source of the enterogenic origin of the disease, various measures have been proposed for cleansing the bowels which are to be an improvement over the administration of cathartics.

Herter favored the method of colonic irrigation with normal salt solution and has reported some very gratifying results by this means, noting particularly that coincident with the improvement, the anaerobic bacteria became less in the stools. He drew the natural deduction from this fact, that there was some etiological relation of these organisms to the disease process.

Recently an operative procedure has been proposed known as appendicostomy, which has been strongly endorsed by Dr. J. A. Witherspoon of Nashville in reporting a case before the last meeting of the Mississippi Valley Medical Association at Louisville, Ky., last October.

A patient was admitted to the University Hospital a few months ago who seemed to be an adaptable case for applying this method of treatment, so that the experience of this one case may be of interest to report herewith. The history is as follows:

Case VI.—*Clinical diagnosis: pernicious anæmia—metaplastic type; hyperæsthesia, paræsthesia, palæsthesia; duration of illness one year; farmer, 54 years of age. Treatment: appendicostomy.*

Mr. S., referred by Dr. Bittner of Wheatland, Iowa. Admitted to the University Hospital May 13, 1909, complaining of stiffness and numbness of the feet and hands, and general weakness. The patient is a farmer, 54 years old, married, and father of five children. Moderate user of alcohol, with no history of venereal infection. Former health good. Present illness of one year's duration, beginning with feeling of tingling and numbness in the feet, which gradually became more marked, and was succeeded by a weakness in the legs which interfered with walking. Only during last month has a pallor been noticeable about his appearance, and he has kept at his work until two weeks ago, when a physician was consulted for the first time. No diarrhœa, moderate constipation; no hemorrhages.

Present Condition.—Very anæmic appearance, yet apparently well nourished; scleræ icteric, skin lemon tint; heart and lungs normal; abdomen slightly distended; liver and spleen not palpable.

Extremities.—Arms, gross strength good, no ataxia, reflexus normal. Legs, gross strength diminished; knee-jerk present; no ankle-clonus or Babinski reflex. Sensation: tactile sense diminished; pain sense increased; has disturbance in temperature sense, especially to heat. Unable to recognize vibrations produced by the tuning fork from the knees downward. The gastric analysis reveals an absence of free hydrochloric acid. Urine is negative.

The *blood findings* upon admission were: hæmoglobin (Dare) 42 per cent.; red cells 1,890,000; leucocytes 2400; marked poikilocytosis, microcytosis, macrocytosis and some polychromasia.

The matter of an appendicostomy operation to facilitate the irrigations of the bowels, was suggested to the patient and upon his consent, it was performed by my colleague, Dr. Jepson, on May 18, the cæcal end of the appendix being attached to the abdominal wall, and a medium sized catheter being fixed in the appendix lumen. After three days the irrigations of the colon were begun by using 1500 c.c. of normal salt solution twice daily. He remained in the hospital until June 24, when he was referred to his home physician, who continued the irrigations.

On August 4 the patient returned and remained one week, after which he was referred back to his physician. His improve-

ment was very manifest in his general appearance, ability to walk better and change in the blood-findings. The paræsthesia and hyperæsthesia were better, but the palæsthesia was the same as before. The achlorhydria remained unchanged. The change in the blood is best illustrated by the following outline:

Fig. 5.

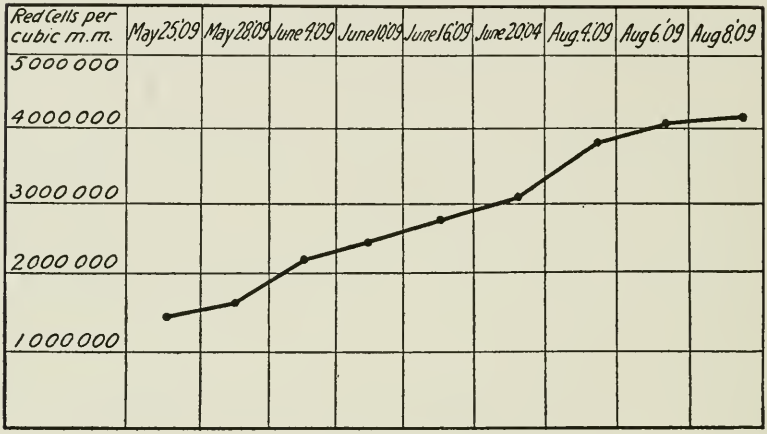

Chart of Case VI.

In contrast to the above case I beg to submit the history of another patient who was under observation about the same time and in which no special form of therapy was applied.

Case VII.—*Clinical diagnosis: pernicious anæmia; blood findings of metaplastic type; chronic diarrhœa; duration of illness eight months.*

Miss S. G., Iowa City, admitted University Hospital May 13, 1909, complaining of general weakness and shortness of breath. Patient is a single lady, housekeeper, 60 years of age. Family history negative; former health good. During the past winter had an attack of influenza after which gradually lost strength. Became very weak and felt compelled to give up her work about two months ago. Moderate degree of diarrhœa and occasional attacks of vomiting prevailed during this time. When admitted patient was very pale, yet the general nutrition did not seem to be greatly affected. Aside from a hemic systolic murmur over the præcordium, the general physical examination did not reveal any-

thing abnormal. The gastric analysis revealed an absence of free hydrochloric acid. The urinary findings were negative. Nervous disturbances were not present.

Blood.—Hæmoglobin (Tallquist) 60 per cent.; red cells 1,320,000; leucocytes 1600; differential count (200 cells), polymorphonuclear 56 per cent., large mononuclear 8 per cent., lymphocytes 32 per cent., mast cells 4 per cent., nucleated red cells 2 per cent. Marked poikilocytosis and polychromasia.

The *treatment* consisted of moderate rest, a nutritious diet in which nitrogenous foods were somewhat restricted; Fowler's solution in doses of three to five drops three times daily, and colonic irrigations of 1000 c.c. of normal salt solution once daily.

The diarrhœa has persisted, the patient having from two to three loose stools each day, but otherwise the patient has undergone a steady improvement, which is best shown by the following chart of the blood curve:

FIG. 6.

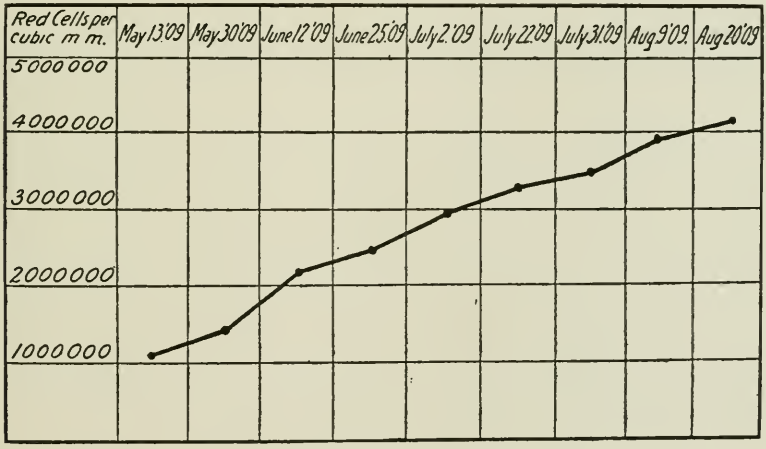

Chart of Case VII

It will be noted from this, that the improvement was equally as good as in the case where an appendicostomy was performed, even though a better method of colonic irrigation was more desired by reason of the greater prominence of the intestinal symptoms.

Admitting that the operation greatly facilitates the cleansing of the large bowel, I question whether it is to be generally recom-

mended in view of the results thus far obtained. At the age when pernicious anæmia usually develops, the appendix has undergone considerable atrophy, so that the operation is more of a cæcostomy rather than an appendicostomy. Aside from the annoyance peculiar to a persistent intestinal fistula, is the discomfort attending the daily irrigations through the fistulous opening, which are necessary in this plan of treatment.

Since the element of hæmolysis enters prominently into the production of pernicious anæmia, it is but natural that remedies should be suggested with the purpose of counteracting the excessive lysis of the red cells. Of these cholesterin has been frequently mentioned, but the experience attending its somewhat limited application in this condition, hardly justifies its continuance as a therapeutic measure. Of the different medicinal measures arsenic is used in the vast majority of cases, although the manner of its action is not understood. It is used generally in the form of Fowler's solution in doses varying from 2 to 10 and 15 drops three times daily, being kept up for weeks and months.

Formerly it was thought that the ability of the patient to withstand arsenic was in a sense a prognostic criterion, *i.e.,* if he bore it well, the prognosis was better than if unable to do so; yet in the study of the remissions it is noted that improvement takes place with or without arsenic. There appears to be no advantage in the use of atoxyl, sodium cacodylate or subcutaneous application of arsenic preparations over the older methods.

Ingestion of bone-marrow, inhalations of oxygen, and various other measures have not withstood the test of time, and it is difficult to believe that any drug, even arsenic, has any considerable influence over the course of the disease.

Patients affected with pernicious anæmia, owing possibly to a lowered psychical balance, are not easily controlled by the physician, and as a rule do not submit cheerfully to any prolonged course of treatment. They become an easy prey to the allurements of the Christian Science "Healer," the attractions of goat-lymph cures, etc. and the manipulations of the osteopath; if then an interval of improvement should opportunely occur, the healing measure applied at the time will receive all the credit for the apparent cure.

At best a remission is but a temporary improvement, and the practical point that suggests itself is the need of reserve in prognosis, and caution in interpreting the results of various therapeutic measures, as the most promising are frequently unstable, meaning an amelioration more often than a definite cure.

If, in the light of present knowledge, a general outline of treatment were to be proposed, it should include (a) a judicious application of rest; (b) a nutritious diet in which the food stuffs rich in purin bodies are to be restricted; (c) a course of arsenic in moderate doses; (d) some measure, preferably colonic irrigation, to facilitate the cleansing of the large bowel, and (e) encouragement, for this is a diseased state in which some of the methods of psychotherapy can be applied with benefit.

In Cabot's collection of 1200 cases, there are six reported recoveries, which, while it demonstrates the possibility of an occasional recovery, yet also illustrates the frightful mortality of the disease under present methods of treatment. Although our knowledge of the nature of the disease is clearer, and the plan of treatment consequently more rational, nevertheless, while the present lack of etiological unity prevails, it is evident that the finding of a definite curative measure is a problem still to be solved.

ADRENAL INSUFFICIENCY

BY EMILE SERGENT, M.D.
Physician to the Paris Hospitals

In 1855 a physician named Addison published the description of the morbid complexus that bears his name, and demonstrated the co-existence of lesions of the adrenals with the prostration, vomiting, pain, and particularly, pigmentation, that accompany this disorder. The following year Brown-Séquard, the physiologist, observed that animals from whom the adrenals are removed succumb in a short time, proving that these organs are indispensable to life From that moment clinical medicine and experimental physiology combined their efforts, and have, little by little, created an entire new chapter of pathology which no physician can afford to ignore.

It is not my intention to pass in review the entire pathology of the suprarenal glands. I think that I can be more useful by concentrating attention on a definite portion of it which dominates the entire pathology of these bodies. I refer to adrenal insufficiency, and shall confine myself entirely to the means whereby this condition can be detected and treated in a patient.

Adrenal insufficiency can be defined, in general terms, as the diminution or suppression of the suprarenal function; it holds the same relation to the adrenals as hepatic insufficiency does to the liver. It reveals itself in clinical medicine by symptoms quite similar to the effects of experimental removal of the adrenal bodies; these symptoms combine themselves in such a way as to form a certain number of conditions, whose evolution follows two principal types: slow adrenal insufficiency, and acute adrenal insufficiency. In order to be able to detect adrenal insufficiency in patients a knowledge of these symptoms and states is indispensable, as they are the elements of the diagnosis.

Lesions of the adrenal capsules give rise to two kinds of symptoms: the first are the result of irritation of the nervous plexus about the capsules, and consist, for the main part, in lumbar pain,

radiating in different directions throughout the abdomen, and in the generalized pigmentation of the skin and mucous membranes that characterizes Addison's disease and that I shall mention later on in connection with the diagnosis; the others are the special signs of adrenal insufficiency, depend directly on the suppression of the adrenal function, and can be divided into four main categories: circulatory, digestive, toxic-nervous, and general disturbances.

1. *Circulatory Disturbances.*—The pulse is small and unsteady; arterial hypotension is the rule; it is more or less marked according to the stage of the disorder; it varies, also, from one day to the next, according to circumstances and to whether the patient remains quiet or undergoes fatigue; it is sometimes very pronounced, giving a tension of only 70, 80 or 90; it is the most evident clinical expression of the decrease in the toni-vascular function of the adrenals. This arterial hypotension is accompanied by a symptom called the suprarenal white line, to which great value should be attached in diagnosing adrenal insufficiency. This white line is, in a certain way, the reverse of the meningitic red line. To bring it into evidence the skin of the abdomen should be lightly rubbed with some blunt instrument, or with the pulp of the finger, without scratching and without much pressure; after a few moments a broad white line will be seen where the finger passed along, and this line will gradually become more marked, then remain stationary for three or four minutes, and afterwards disappear little by little. The diagnostic value of this white line has been denied by some writers, but I must say that I have not been convinced by their arguments, and still am of the opinion that this white line is very rare with persons whose adrenals are in a healthy condition.

Together with this arterial hypotension there is almost always tachycardia, a sensation of cold, sometimes very pronounced, and a tendency to collapsus; in many cases the disease ends in syncope; sudden death is extremely common among patients with disorders of the adrenals.

2. *Digestive Disturbances.*—Anorexia is almost the rule; it is sometimes absolute, when the asthenia is very marked. Vomiting is one of the most frequent symptoms. When the disease assumes an acute form this symptom may be quite incoercible and,

together with the diarrhœa and violent pain, may lead to a diagnosis of poisoning. Stubborn constipation generally accompanies chronic adrenal insufficiency; diarrhœa, on the other hand, occurs in the acute forms.

3. *Toxic Nervous Disturbances.*—These reveal the general intoxication of the nervous system. There may be general pain, similar to the cramps of cholera patients, appearing more particularly in cases of acute insufficiency, and quite distinct from the lumbar pain radiating throughout the abdomen. Or there may be cerebral symptoms, chronic, subacute or acute. The chronic and subacute symptoms consist in a condition of partial somnolence, either quiet or disturbed, and mingled with dreams or nightmares, ending sooner or later in coma. This cerebral condition is an element of the asthenia, to which we shall refer presently. The acute variety may appear suddenly without warning, or else manifest itself during the course of a chronic case of insufficiency. It is characterized by violent headache accompanied by agitation, convulsions and delirium, and ending generally in profound prostration quickly terminating in death. There is, finally and above all, a state of asthenia which is, beyond question, the predominant symptom of adrenal insufficiency. Everyone who has seen the asthenia of a patient with Addison's disease will always remember the pitiable appearance he presents. Apprehensive of every effort, which on the other hand he would not be able to furnish, conscious, but with no longer the strength to wish or to act, the patient is an inert object deprived of all physical and moral activity. Wrapt in a permanent condition of somnolence constantly buried under the bed-clothes, he replies only in feeble tones when spoken to, and accepts reluctantly the food brought to him, so great is his dread of the fatigue caused by any slight effort. This condition of profound apathy indicates the approaching disappearance of the suprarenal function, but it is preceeded by a period of greater or less duration during which the patient experiences an increasing sensation of fatigue and lassitude. In some instances this lassitude is so slight that it does not suffice to keep the patient from his daily occupations, but, on the other hand, it can be suddenly superceded by a state of violent depression, that comes on the patient in a condition of apparent health, and attains its

maximum almost at once. This is what takes place in cases of acute adrenal insufficiency.

4. *General Disturbances.*—These consist in a slowing of tissue interchange, with lowered temperature and more or less marked alteration of the patient's health. There is a state of anæmia, of more or less rapid loss of weight, and of progressive cachexia.

It was remarked above that adrenal insufficiency could assume two chief types: one in which the evolution of the symptoms was slow, and the other in which this evolution was rapid; again both of these types comprise a certain number of variations. Let us go over them rapidly, and show how they can be detected and distinguished from the different disorders that they resemble.

1. *Chronic Forms.*—The symptoms of chronic adrenal insufficiency can be considered as the clinical manifestation of progressive diminution of the adrenal function. They bear the same relation to chronic lesions of the capsules as the initial signs of chronic hepatic insufficiency do to chronic hepatitis. This course tends toward final acute insufficiency. They may take two forms: either that of an ordinary case of Addison's disease, or that of pure chronic adrenal insufficiency, which are distinguished by the fact that the first consists in a combination of the signs of adrenal insufficiency with those of irritation of the pericapsular nervous plexus.

Addison's disease, therefore, is a compound state, in which adrenal insufficiency plays a part no doubt important but by no means exclusive. The sign which characterises Addison's disease is the pigmentation, which is absolutely obligatory. Now among all the varieties of adrenal insufficiency Addison's disease is the one that can most readily be recognized, on account of the pigmentation, provided the physician is familiar with its peculiarities. This pigmentation is seen not only in the skin but in the mucous membranes, and has a marked predilection for regions that are pigmented in a normal state, such as the nipples and genital organs. It may be very limited, and consist only of a few brownish spots scattered here and there; or else it may be very pronounced and appear over the whole surface of the body. The tegument first assumes a dirty grayish tinge, then gradually it turns brown,

so that the patient may resemble a mulatto, and in some cases even attains an intensity at which it resembles bronze. Repeated rubbing of the skin, diverse varieties of irritation, and scars, give rise to a specially intense localization of the pigmentation of Addison's disease. This peculiarity also supplies us with a means of diagnosis in cases where there is hesitation, as the mere application of a mustard plaster will give rise to pigmentation when the disease has not yet made itself manifest on the external surface of the body.

If to this sign be added the presence of patches of pigment in the mouth or on the conjunctiva, the physician will be in possession of the chief elements for a differential diagnosis of Addison's pigmentation.

It will hardly be necessary to pass in review the other kinds of skin pigmentation. I will merely say that certain vagabonds have a generalized coloration of the skin which, when combined with their state of physical and moral degeneration, resembles in many points the pigmentation of Addison's disease. It is also well to bear in mind the pigmentation of certain advanced cases of tuberculosis, whether of arsenical origin or not. The consumptive and cachectic state of these patients must be clearly distinguished from pure asthenia. Although Addison's disease is generally based on tubercular lesions of the adrenals, the fact that in these cases there is tuberculosis does not naturally lead to a diagnosis of Addison's disease, because tuberculosis of the adrenals is almost always primitive and is very rarely observed in advanced tuberculosis.

We now come to the diagnosis of the chronic forms of pure adrenal insufficiency. These have been called abortive or partial cases of Addison's disease, but, as will have been seen from what was said above, these terms embody an inexact conception of the anatomical localization of the disorder.

With these patients the diagnosis is more difficult, as the symptom that usually attracts attention, namely pigmentation, is lacking; and yet a diagnosis is possible if the physician is firmly imbued with the idea that the symptom asthenia has at least an equal value as a sign of capsular lesion. Asthenia more or less marked, arterial hypotension with or without white line, anorexia,

constipation, vomiting, anæmia, and progressive loss of weight, constitute a group of symptoms that should awaken suspicion. And yet even here it is well to keep the patient under observation, and to eliminate other disorders first that have analogous symptoms, such as progressive pernicious anæmia, and leukæmia, though with these disorders a blood test will soon clear away doubt. Pulmonary tuberculosis and latent cancer must also be eliminated. As regards tuberculosis, if there are marked physical signs and the case is an advanced one, it is hardly necessary to think of the adrenals, since tuberculosis of those organs is generally primary, or at any rate fairly well localized to the adrenals.

In any case no harm is done by submitting the patient to an organotherapeutic test, which, by means of the results that it may give, will in some instances confirm or annul your presumptions, as I shall mention later on.

2. *Acute Forms.*—Acute suprarenal insufficiency corresponds to sudden diminution or suppression of the suprarenal function. Its symptoms bear the same relation to acute or chronic suprarenal lesions as those of acute hepatic insufficiency do to acute or chronic hepatitis. It is characterized by a combination of symptoms that are in a general way a mere exaggeration of the symptoms of chronic insufficiency, but which, on the other hand, unite and pass through their phases with a rapidity that is sometimes fulminating.

The forms of acute suprarenal insufficiency may appear secondarily during the course or toward the end of a chronic case, or else suddenly in patients apparently in ordinary health.

When they appear secondarily they are not difficult to diagnose. This is the case with the acute symptoms resembling an intoxication that are so common in Addison's disease. They may even in some cases confirm a diagnosis that has been wavering. When during the course of chronic insufficiency without pigmentation sudden symptoms set in resembling poisoning, the idea of suprarenal insufficiency will come to the mind if the physician has heard of these instances, whereas otherwise he might not have thought of this possibility.

When they appear suddenly in a patient in apparently good health, they take the physician unawares and lead to a variety of errors. For what sign should lead him to the diagnosis, if he

is not familiar with such cases? The attack starts almost always with severe pain in the back and abdomen, and with generalized cramps that cause the patient to cry out. It is accompanied by uncontrollable vomiting, with or without diarrhœa, the skin is covered with cold sweat, the extremities are cold, the temperature falls, the pulse grows weak, arterial tension decreases, the white line appears, and if the patient does not pass into collapse or coma, he succumbs suddenly during a paroxysmal crisis, or simply on sitting up in bed during an interim of quiet.

It will be seen that this is the picture of an ordinary case of poisoning, and this is what the physician generally thinks of, whether the poisoning be voluntary, criminal or accidental. These cases have the greatest importance, not only from a clinical point of view but also from a medico-legal aspect.

In other cases the symptoms resemble rather those of the acute abdominal conditions, or of an attack of appendicitis, or else a case of dry cholera. In other instances the cerebral manifestations predominate and the evolution of the disorder follows the type of meningitis, or may even be taken for an attack of apoplexy.

It will be readily seen how difficult the diagnosis may be with such patients. Still, it is not impossible and I was once even able to make the diagnosis where all the symptoms pointed to meningitis. This, however, I will admit was due to the fact that at that time my attention was closely concentrated on researches concerning suprarenal insufficiency, otherwise it is not likely that I should have thought of this disorder. For as a general thing, and so long as our minds are not familiarized with these new data, the proper diagnosis will not be made and the error will only be rectified at postmortem. Still, without wishing to exaggerate in any way, it can be said that whenever such symptoms cannot be traced to a manifest cause, whenever their nature cannot be demonstrated at once and beyond question, we are authorized in raising the point whether they may not be due to acute suprarenal insufficiency.

As a help we can have recourse to certain etiological data, which in my opinion, play an important part as elements in the diagnosis of acute suprarenal insufficiency.

Thus, any traumatism may give rise to an attack of acute

insufficiency in a patient with capsular lesions latent up to that time. This origin is mentioned in several cases; it was either an accidental fall, an assault, or the shock of an operation. A case of the latter variety has been published, in which a woman succumbed rapidly from acute insufficiency after an operation on the uterine appendages. Facts of this kind may have considerable importance in legal medicine.

But it is, above all, the infectious diseases that are capable of giving rise to the condition of acute suprarenal insufficiency, whether in patients with chronic capsular lesions in course of evolution, or in patients whose suprarenals had up to that time been in good condition. In the former instance the mechanism is complex, and it is uncertain whether this insufficiency, latent up to that time, is due to additional acute lesions of the suprarenals, or whether it is only the result of the inability of the damaged suprarenals to accomplish an unusual antitoxic strain. It is likely that the latter mechanism is the true one, when the symptoms of acute insufficiency occur on the occasion of some slight infection, such as a simple sore throat, as has been reported several times.

In the latter instance the infectious disease, by giving rise to acute suprarenalitis, produces suprarenal insufficiency. Suprarenal localizations are very common in the more important toxi-infectious disorders. I have spent a great deal of time over the clinical study of these localizations, and have had the satisfaction of seeing my views adopted and confirmed by a certain number of authors, particularly in connection with scarlet fever and diphtheria. Typhoid fever, pneumonia, the other infectious disorders, and even mumps, may have a similar result.

It is evident that in these cases the symptoms of acute insufficiency are less clearly defined, because they are, as it were, swamped by the symptoms of the toxi-infectious disorder during which they appear.

For my part I am of opinion that when in the course of an infectious disorder there suddenly appears a state of depression, with temperature below the normal, and arterial hypotension with white line, which follows a former acute febrile state with fever and hypertension, the physician is justified in suspecting

that the suprarenals are affected, and that the lesion is sufficiently serious to interfere with their function and threaten the patient's existence. This fact is of great practical importance, as it is evident that it is particularly in cases of this sort, where the lesion it not a fatal one, that suprarenal organotherapy may have a chance of being efficacious, to which point we shall refer in a moment.

From all that has been said above there follows, in short, an important precept, that the attention of physicians cannot be too strongly directed to the existence of pure suprarenal insufficiency, chronic or acute. It is desirable that they should be fully impregnated with the idea that Addison's disease does not comprise in itself the entire pathology of the suprarenals; that the pigmentation is neither necessary nor sufficient for the diagnosis of a capsular lesion; that the asthenia is at least fully as valuable a sign; that acute primitive accidents resembling poisoning, peritonitis, meningitis or apoplectic coma, are, in some instances the sole expression of suprarenal insufficiency; and that, finally, this condition is not rare during the course of infectious disorders, in which its existence is to be suspected when the acute febrile symptoms of the early stages are suddenly succeeded by signs of depression, subnormal temperature, and arterial hypotension.

No doubt the diagnosis remains uncertain in the majority of cases in spite of the signs of strong presumption that may exist. It is for this reason that I have endeavored to find a symptom to a certain degree pathognonomic of suprarenal insufficiency, and think that I have found it in what is called the suprarenal white line. Nor do I feel that the criticisms that have so far been made of this symptom have at all done away with its value.

Although a knowledge of suprarenal insufficiency is important in clinical medicine, it is equally so in legal medicine, as I have mentioned above. It should always be borne in mind that capsular lesions frequently produce sudden death, and that they also reveal themselves by acute symptoms similar in every way to those of poisoning. It is desirable, finally, to be well acquainted with the fact that the signs of suprarenal insufficiency can be suddenly revealed by accidents, operations, infectious diseases, and even pregnancy, in patients with capsular lesions in evolution or latency.

The treatment of suprarenal insufficiency comprises preventive and curative measures.

By preventive measures are meant those designed to prevent the occurrence of symptoms of acute suprarenal insufficiency in patients suffering from a slow form, whether Addison's disease, or slow insufficiency without pigmentation. With these patients the suprarenal function is considerably diminished. It is still sufficient for work at low pressure, but it will fail if the muscles are freely used, or if an infectious disorder or intoxication intervenes. These patients must therefore abstain from all fatiguing work, avoid contact with patients suffering from infectious diseases, refrain from toxic drugs, and particularly from arsenic which is a violent poison for the suprarenals, and only submit to an operation if it is rigorously indispensable. On the other hand, they would derive benefit from a phosphoric treatment, and particularly from Lecithin.

The curative treatment comprises, in particular, the organotherapeutic medication. But it is first necessary to refer to the specific treatment for syphilitic patients presenting symptoms of suprarenal insufficiency, for a certain number of cases of recovery of Addison's disease through mercurial treatment have been published. But mercury, and even iodide, must in such instances be handled with the greatest precautions, as they are violent poisons for the suprarenal capsules.

But with the exception of these special cases, which are very unusual, the treatment of suprarenal insufficiency is by organotherapeusis. A detailed examination of this method will be found in the publications of the last few years, and particularly in the monograph on suprarenal insufficiency written by me in collaboration with Bernard. For the time being I will limit myself to purely practical indications.

It is unquestionable that suprarenal therapy effects considerable improvement in patients suffering from suprarenal insufficiency. In this connection there is apparent disagreement between former cases and more recent ones, due, I think, to the fact that suprarenal extracts are better prepared nowadays. I myself had occasion last year to follow a case of Addison's disease in which, under the influence of the suprarenal treatment, I saw a most pronounced asthenia improve considerably, while at the same time the arterial hypotension and the white line disappeared.

The suprarenal treatment can be given in several ways. It was tried in the first place in the form of injections of glycerine extract, but in addition to the fact that these are very painful, they often give rise to abscesses, and have been gradually abandoned. Nowadays we administer it by the mouth, either in the form of the fresh gland, or of dried extract. The fresh gland should be procured from the slaughter house every day from young calves. The initial dose should be from one and a half to two grammes, to be increased gradually to five grammes, or even more. As a matter of fact, however, it is no longer as necessary as it was a few years ago to use the fresh gland, since we now have at our disposal a certain number of very good pharmaceutical preparations of dried extract. If the wafers prepared by Carrion, containing 30 centigrammes each, be used, three should be given per diem, and the pulse and stomach of the patient closely watched. This should be continued for ten or twelve consecutive days, and afterwards resumed after an interruption of two or three days.

In the majority of cases it is advantageous to administer the total extract; still, in some instances adrenalin can be given, particularly in treating a patient with an infectious complaint in whom a suprarenal localization is suspected. For several years now it has been my custom to administer adrenalin whenever signs of cardio-vascular asthenia appear in patients suffering from infectious diseases. I have been able by an intermittent use of this drug to convince myself that the suprarenal white line appears and disappears according to the oscillations of the arterial tension, which has seemed to me an argument in favor of the interpretation which I have given of this phenomenon.

When, under these conditions, recourse is had to adrenalin, the daily dose should be from 20 to 30 drops, that is to say, from one to one and a half milligrammes. This can be raised up to five or six milligrammes per diem by spreading this quantity out into five or six doses during the 24 hours. The administration of adrenalin can be continued for two months, at these doses, and with these precautions, without fear of producing atheromatous lesions of the vessels.

THE PROGNOSIS IN ADVANCED TUBERCULOSIS

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PHILADELPHIA

A REVIEW of the present medical literature will show many articles dealing with tuberculosis from the clinical standpoint. The vast majority are upon the diagnosis and treatment of early or incipient cases. While there can be no question as to the necessity of early diagnosis the fact remains that there are at present a large number of advanced cases which require treatment, and that these advanced cases make up a large proportion of the cases of tuberculosis which come under the care of the general practitioner. My own experience shows that the early cases are rather hard to find while the more advanced ones are plentiful.

The statistics of the White Haven Sanitarium show that out of 1780 patients admitted during the period between August, 1901, and February, 1907, the *incipient cases* were 24.04 per cent.: moderately advanced, 40.39 per cent.; far advanced, 35.56 per cent. Thus, more than 75 per cent. of the patients admitted to a sanitarium for early cases were beyond the incipient stage.

The important fact is that while we have a large number of advanced cases of tuberculosis to be treated, our medical journals discuss only the care of the incipient case. This tends to emphasize the already pessimistic view of the general practitioner regarding the possibilties of the advanced case and takes from his treatment the enthusiasm and optimism so necessary in the management of tuberculosis at all stages. Is this pessimism justified?

The statistics of the results on discharge at the White Haven Sanitarium in patients who stayed longer than three months are:

MALES

STAGE 2.—MODERATELY ADVANCED

Disease arrested	149	(40.72%)	; av. gain in weight, 20.00 lbs.
Very much improved	29	(7.92%)	; av. gain in weight, 19.00 lbs.
Much improved	80	(21.86%)	; av. gain in weight, 17.50 lbs.
Improved	89	(24.32%)	; av. gain in weight, 12.90 lbs.
Not improved	18	(4.91%)	; av. loss in weight, 4.64 lbs.
Died	1	(0.27%)	; av. gain in weight, 18.00 lbs.
Total	366		

STAGE 3.—FAR ADVANCED

Disease arrested	23	(26.75%)	; av. gain in weight, 17.63 lbs.
Very much improved.	4	(4.65%)	; av. gain in weight, 23.00 lbs.
Much improved	22	(25.58%)	; av. gain in weight, 11.15 lbs.
Improved	34	(39.54%)	; av. gain in weight, 11.75 lbs.
Not improved	2	(2.32%)	; av. gain in weight, 0.50 lbs.
Died	1	(1.16%)	; av. gain in weight, 0.00 lbs.
Total	86		

FEMALES

STAGE 2.—MODERATELY ADVANCED

Disease arrested	110	(44.0 %)	; av. gain in weight, 00.00 lbs.
Very much improved.	24	(9.6 %)	; av. gain in weight, 21.63 lbs.
Much improved	54	(21.6 %)	; av. gain in weight, 24.95 lbs.
Improved	51	(20.4 %)	; av. gain in weight, 21.41 lbs.
Not improved	9	(3.6 %)	; av. gain in weight, 14.00 lbs.
Died	2	(0.8 %)	; av. gain in weight, 3.90 lbs.
Total	250		

STAGE 3.— FAR ADVANCED

Disease arrested	19	(38.90%)	; av. gain in weight, 20.61 lbs.
Much improved	14	(28.50%)	; av. gain in weight, 14.37 lbs.
Improved	12	(24.44%)	; av. gain in weight, 15.12 lbs.
Not improved	4	(88.16%)	; av. gain in weight, 2.81 lbs.
Total	49		

These results prove very conclusively that much can be done for the advanced case. Life can be prolonged, suffering diminished and in many cases the patients can perform useful work, particularly when his occupation is one not requiring muscular exertion.

The factors upon which the prognosis of advanced cases may be classified are as follows:

1. Age.—Generally speaking, patients over 35 offer a more favorable outlook. This is particularly true in patients among the well-to-do. There are several factors which combine to make the age of the patient important. Among these may be mentioned the fact that at 35 the average individual has practically established his or her position or occupation in life and it is possible to estimate pretty accurately the amount of energy to be expended in retaining or continuing in this position or occupation.

2. Sex.—Personal experience leads me to believe that the male patient is apt to give a better ultimate result than the female patient.

Women as a rule cannot adapt themselves to the systematic life necessary to the tuberculous patient. An exception to this is met with in women in institutions like hospitals, sanitaria, and convents, where they are more or less under discipline. The especial difficulty lies in the diet. After men are once convinced that some particular diet is necessary and have taken it long enough to prove to themselves that it produces good results they accept it as a necessary accompaniment of their disease and make no further protest. The average woman patient takes each glass of milk and each egg under protest and no amount of proof can make her appreciate that when the appetite is unable to make one take enough food she must bring her will-power into play and of her own accord make up for the deficient appetite. It may be that the reason that milk is so distasteful to women is because women in general seem to have an aversion to liquids. It is astonishing to note the number of women, apparently well, who go all day long without drinking water. Another fact in a womans' life is the monotony of her surroundings when at home. Finally may be mentioned the undoubtedly bad effect of pregnancy in the tuberculous woman together with the bad effect of the recurring menstrual periods. Very many tuberculous women show increased temperature, blood tinged expectoration, and increased râles in the lungs at the menstrual period. In others a distinct increase in the length of the period or in the amount of blood lost may be among the first evidences of a beginning breakdown from tuberculosis.

3. Duration.—All things considered a longer duration of the symptoms of the disease speaks for a greater amount of resistance upon the part of the patient. In other words, if a patient shows involvement of two lobes after symptoms lasting two years there is probably a greater resisting power than in a case of the same amount of involvement with symptoms lasting only a few months. The mode of life of the patient should be taken into consideration in the estimation of this factor as one who had been living in a sanitarium ought to be in better condition than the patient not under treatment.

4. Financial Condition.—This is very important. As a rule the patients who are in the most comfortable circumstances financially have the easiest work and are most intelligent. The very

rich, however, are subject to so many temptations that a strict mode of life is difficult.

5. Incentive.—Besides the desire to live for lifes' sake, if there is also some other incentive the outlook is better. Thus, for the sake of a family, or some other special object, I have seen patients put forth more effort than they would for themselves alone.

The above may be looked upon as general considerations in the prognosis of the individual case. Next comes the more important data which includes the physical examination. Needless to say this physical examination must be complete and made without prejudice upon the part of the examiner. He must remember that the lungs are not the only important organs in the body. He must determine the amount of good tissue as well as the amount of diseased tissue. The heart, abdomen, kidneys must all be studied. In making his prognosis the thought should not be "how much disease has this patient"—but rather, "how much good material have we to work on?" Tissues diseased with tuberculosis do not offer much chance of repair. But it is surprising how much work can be done by a small amount of good, healthy tissue which is properly nourished and properly trained.

Let us now consider the physical examination in detail:

1. *Temperature.*—This should be taken several times a day and for at least 15 minutes each time, before we can say that fever is absent. Very often tuberculous patients will not show a rise in temperature when the thermometer is left in the mouth during the time stated on the instrument. This is most likely due to the lack of blood in the mucous membranes of the mouth which produces the characteristic pallor so common in the mouths and throats of tuberculous patients. It is hardly necessary to mention that temperature taken in the morning is apt to be misleading. High temperature in advanced cases may be due to an intercurrent infection or in women may occur during the menstrual period, and one should not draw conclusions from it alone. Sometimes high temperature is disregarded by the physician who explains it on the ground of nervousness or over-exertion. The fact that either of these can produce a rise in temperature is very important as it shows an unstable equilibrium of the body which requires urgent treatment. In the advanced cases it is always wise to withhold judgment on the temperature until the patient has been in bed.

Normal temperature in the advanced case is a good prognostic sign as it indicates lack of toxæmia.

2. *Pulse Rate.*—Under this is considered the examination of the heart. Great care is to be taken in estimating the condition of the circulation. The pulse should be counted at the wrist or with a stethoscope over the heart with the patient standing, sitting and lying. In this way one is able partially to estimate how much of the rapidity is due to nervousness; how much to toxæmia; how much to physical exhaustion. A rapid pulse means much less in tuberculosis than in almost any other condition. The great majority of the incipient cases which present themselves for examination for White Haven show a pulse rate of 120. A normal or slow pulse rate when at rest speaks for an absence of toxæmia. A distinct lowering of the pulse rate when the patient lies down is an evidence that a portion of the rapidity is due to physical exhaustion. Slow pulses are infrequent. I have one very advanced case whose pulse rate is 44 to 48 when recumbent.

3. *Respiration.*—Rapid respiration when at rest may mean extensive involvement but more often is an evidence of some complication as pleurisy with effusion or a pneumothorax. Occasionally it is due to kidney insufficiency. Rarely it may be due to nervousness. I have one case now under observation whose respiration is 75 per minute while the pulse rate is only 68 to 72.

4. *Urinalysis.*—This should include an examination for Diazo and of the centrifugated specimen under the microscope. A positive Diazo is unfavorable. Albumin is rarely present in large amounts and may be absent even when there are clinical evidences of kidney insufficiency. The microscopic examination is the most important. When casts are found the outlook is bad, especially in young people.

5. *Digestion.*—Inasmuch as our modern treatment of tuberculosis depends largely upon feeding, the condition of the digestive tract is of the greatest importance. Lack of appetite is the rule. Discomfort after taking food is most often due to too much exercise. It must be remembered that the muscles of the digestive system suffer along with those of the rest of the body when the general vitality is reduced. The important digestive symptom is diarrhœa. When this is present there is a tendency upon the part of the physician to assume the presence of intestinal tuberculosis.

As long as there is no enlargement of the liver we are justified in giving the patient the benefit of the doubt. Walsh has shown from a number of cases followed to the autopsy table that diarrhœa may be present without ulceration and that ulcers may be present without pain or diarrhœa. Flick and Walsh pointed out that diarrhœa may be an evidence of kidney failure with a vicarious action of the bowels. Many of these cases with a history of diarrhœa will explain, on closer questioning, that the loose bowel movements occur chiefly in the morning when there is one formed stool followed at rather frequent intervals by several loose movements. These cases usually respond to treatment and I have thought that the loose movements were but one of the evidences of the relaxation so common in the morning in old tuberculous cases. At times, in cases who have been taking milk and eggs a long time, the patient reaches a stage where a bowel movement follows each feeding of milk and eggs. Most of the cases I have seen have been in women and the condition is very difficult to manage. Complete change in surroundings and diet are necessary. So many of the cases of diarrhœa seen in tuberculosis are of nervous origin that a fair trial of treatment should be made before they are considered as hopeless. I have one patient now having normal stools who had a diarrhœa of eight to ten movements daily for three years.

Further Physical Examination.—It may seem unnecessary to say that in every case the patient should be stripped to the waist and carefully examined by inspection, palpation, percussion and auscultation. This examination is of especial value in the advanced case where the diagnosis of tuberculosis is relatively easy by the finding of tubercle bacilli in the sputum. Unfortunately it is in the advanced case that this examination is neglected. The physician contents himself with making a diagnosis of tuberculosis and then prescribes a routine treatment. This failure of a thorough examination is the only possible way to explain the directions for exercise given to active cases with rapid breaking down of the lungs. It is not uncommon to find patients who are steadily losing in weight taking active exercise and deep breathing by direction of the physician when each day so spent tends to hasten the progress of disease. I would like specially to emphasize the necessity of examining every patient in the recumbent position as well as when standing or sitting. Râles which cannot be heard when stand-

ing become very evident when lying down. Just why this should occur is hard to say, but it is a rather frequent finding. As already stated the plan of examining the patient in both positions gives a much better idea as to the condition of the heart.

Without going into details as to the various combinations of lung involvement that are found in advanced cases, I want to call particular attention to one special form of advanced tuberculosis in which the outlook is often favorable although the extent of disease is very great. These are cases with complete involvement of the left lung with moist râles throughout and usually associated with a small quiescent lesion in the right apex. The cases that I have seen have given a history of illness or of failing health over a period of from one to ten years. In only one instance have I had the opportunity to see an early case, that is, within two months of the first subjective symptoms. The older case presents itself with a history of long standing illness.

Has cough, considerable expectoration, loss of weight, dyspnœa on exertion, and all the subjective symptoms of chronic tuberculosis. The physical examination shows the left chest to be smaller and with diminished motion, while the right chest is correspondingly larger and more active. The heart is apparently displaced, the apex beat being outside the mid-clavicular line to the mid-axillary line and in one case could be felt under the angle of the left scapula. Percussion gives tympany or dulness over the upper lobe and impairment or dulness over the lower lobe. Auscultation gives amphoric breathing and pectoriloquy over the upper lobe together with numerous moist and dry râles. Over the lower lobe the râles are so numerous as to mask the breath sounds and one usually fails to notice that the signs of a cavity, such as pectoriloquy, are usually absent. The right lung is hyperresonant and gives this full note often to the left border of the sternum. On the right the breath sounds are harsh, showing that the lung is doing extra work.

While this involvement is very extensive many of these cases are capable of marvelous improvement. Quite often the râles may be entirely cleared from the lower lobe and the patient be restored to normal weight, pulse and temperature. The fact that I want to emphasize is this, that while wonderful and unexpected recoveries occur in these left-sided cases, I have not been able to

find any case with complete right-sided involvement that gave as good results. Certain cases of right-sided involvement have done well, but I have never seen one who was able to get back to a condition of a normal pulse rate.

I have records of a number of these left-sided cases in which the improvement has been very great. In some the lower lobe has become free of râles. In all of my cases signs of disease remain in the left upper lobe. Some of the cases which have regained the normal condition as to temperature, pulse rate, and working ability, still have râles throughout the entire left chest. In one case there is a definite history of these râles being present for the past fifteen years. The great majority of these left-sided cases which do well show a displacement of the apex beat outward. I have seen a few cases in which this displacement did not occur and in those the pulse rate remained high and the working ability proportionately lower.

In attempting to explain this great difference in the right- and left-sided cases a large number of factors must be taken into consideration. It may be possible that the condition in the lower lobe involves largely the pleura and some of these cases do start as pleuropneumonias or pleural effusions. Again it may be considered that the signs over the lower lobe are those of congestion only and do not mean actual disease of this lobe. Chronic congestion of a lung usually means failure of the circulation, and it is hard to realize a condition which would produce a unilateral congestion. The one thing that seems most striking is that cases showing great destruction of tissue on one side do lead to apparent displacement of the heart. When this apparent displacement is to the left the circulation can regain an apparently normal condition, while when the apparent displacement is to the right the heart is always embarrassed. The most plausible explanation of this difference in results of the two types of cases seems to be as follows.

Return to health and strength after marked destruction of one lung requires the enlargement of the remaining lung and a consequent readjustment of the thoracic relations. The enlargement of the one lung and diminution of the size of the other bring about changes in the thoracic pressure relations. When the left lung is partially destroyed the vacant space left is partially filled by the falling in of the chest, partially by elevation of the diaphragm,

partially by the extension of the right lung towards the left. Under these conditions the apex beat changes its position and is found far outwards in practically the same position as when the normal individual lies on the left side. There is really almost no actual displacement of the heart and therefore no mechanical interference with the circulation.

When the right lung is affected and to a great extent destroyed, the readjustment is less easy. The diaphragm is less easily elevated because of the liver, and there is a constant pull on the heart toward the right. Displacement of the heart laterally is prevented by the inferior vena cava which is practically a component part of the diaphragm and liver. Under these circumstances the circulation is more embarrassed because of the tension placed on the inferior vena cava.

In other advanced cases the circumstances under which the examination is made may bias the examiner's judgment. Occasionally, patients are first seen after a long journey on a train or after other unusual exercise. Under these conditions the pulse rate is much higher and the number of râles and the extent of lung over which they can be heard is greatly increased. Rest in bed for a week or two often shows a marked diminution in the pulmonary signs and gives one a more favorable conception of the case. Even when the râles persist for a longer period of time, if the course of the temperature and the gain in weight is satisfactory we can still hope for improvement.

The point I wish to emphasize is this. Because a case of tuberculosis is advanced we have no right to give a hopelessly, unfavorable prognosis. Prolongation of life and relief from pain and weakness is all that such cases wish for. It is a mistake to think that because such a patient cannot be restored to complete strength that he wishes to die. We are all convinced of the necessity of not promising too much in the way of cure, and we should be equally careful of our promises in the other direction. Many of us are inclined to be proud of being "honest" with a patient and translate the word "honest" to mean putting the most favorable interpretation upon all the signs found. What should be done in giving a prognosis in advanced tuberculosis is to study the case carefully while under proper treatment. It is only then that we are justified in giving a hopeless prognosis.

GOUTY PHLEBITIS*

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Gentlemen: If you look in any of the best treatises on medicine that were published fifty years ago you will not find any mention of the condition which I propose to discuss to-day. Attention was first distinctly directed to the variety of phlebitis which is now recognized as of gouty nature by Sir James Paget.[1] The older writers appear to have regarded phlebitis, as ordinarily met with, as more or less directly connected with pyæmic states, and mainly dependent on injuries demanding surgical rather than medical treatment. It is curious to find that the particular variety we are to consider to-day was so long in being recognized as a manifestation of a gouty habit of body.

Systematic writers describe as the main varieties of phlebitis, an adhesive and a suppurative form. The former is commonly local and of traumatic origin, and may pass into the latter condition, becoming diffuse. The symptoms vary much according to the position of the involved veins whether they be superficial or deeply seated. We are not concerned with the suppurative variety which may arise from a septic thrombus at a distance, or from an adjacent local venous plug, and in the gouty form we are not apt to meet with any but the adhesive variety. As you are aware, suppuration forms no part of a strictly gouty inflammatory process. Diffuse suppurative phlebitis is a grave disease of septic quality with general infection, sometimes leading to gangrene, and often to death by septicæmia or pyæmia. The pathogenesis of phlebitis may be clearly classified in two categories, (*a*) the *infectious* varieties, and (*b*) the *constitutional* varieties. In this lecture we are concerned solely with the latter in one of its most marked forms.

The subjects of gouty phlebitis are commonly of the male

* A lecture delivered at the London School of Clinical Medicine, Seamen's Hospital, Greenwich.

[1] St. Barth. Hosp. Reports, 1866.

sex, and in middle or late life. The lower extremities are the usual sites. The patient is either obviously a gouty subject, or is distinctly prone to some manifestation of the gouty habit. The first symptom is that of local pain with slight itching, generally in some branch or tributary of the saphena vein. There may be severe pain in some cases at first. On examination, there is found some slight redness over the affected part, and a distinct cord is to be felt in the involved branch, tender to the touch. In slight cases, there is nothing further to be recognized. If there are several branches involved, or if a large vein is the seat of the process, there will be some degree of œdema of the limb below the part. The gravity of the case depends on the size and amount of venous structure implicated, and it is always more serious to find deep-seated veins involved than those which are superficial. In the former case, the cord-like inflamed veins may not be detectable, but the degree of œdema is a fair criterion of what has occurred. In mild cases there may be no pyrexia, and no marked failure of health. The patient may prove to be a free liver, perhaps generally plethoric, and may have been indulging previously in too many of what are termed "the pleasures of the table." In addition to this he may have been taking strong exercise, hunting or riding or over-walking. I have known the friction of stirrup-leathers to set up phlebitis in the calf of the leg in such cases, when riding boots have not been worn, and the extra exertion of walking along slippery, muddy pavements or snow has also been a provoking cause in my experience. Note, that in all such cases the patient is in a special gouty state at the time, and ready for an outbreak at some point, articular or abarticular, that is, at a *locus minoris resistentiæ* as the old writers would have described it.

It is a question of degree as to the numbers of veins involved, and also, as regards the severity of the process in question, as to superficial or large and deeper veins being involved. The particular locality, too, may, obviously, affect the prognosis in any case. One peculiar feature of gouty phlebitis is a tendency for the process to occur in patches at the same time, without any direct connection between them, and to affect different parts of the body.

It has long been a problem in pathology as to the order of events in any case of phlebitis; that is, whether the first error in the process concerns (*a*) the contents of the vessel, or (*b*) the walls of the vessel. By the time the ailment is detected there are indications generally of both of these conditions, viz., a clot in the lumen (thrombosis), and an inflammation of the tunics of the vessel (phlebitis proper). In any case, by the light of modern teaching we may feel sure that there is a toxic process at work whether it be an example of direct traumatism, or of pyæmia, or, as in the instance before us, of gout. So far, all is clear. In the two former conditions we may be sure of some microbic invasion, but we have as yet no evidence of this in any truly gouty process, albeit we have to deal with an autotoxæmia so far as we know, engendered by perverted systemic metabolism, roused into activity for the time being. And, as we have seen, there is in this case commonly a traumatic element or a direct irritation set up by friction or over-strain.

I have already alluded to the patchy nature which is apt to pertain to gouty phlebitis, and there is sometimes a symmetry to be noted in these separate patches which may occur at intervals. In this quality we may note a similarity to attacks of ordinary gouty inflammation which may alight on various parts of the body, not always articular, in the course of one attack. This peculiarity led Sir James Paget to believe that this feature of the ailment was significant rather of an inflammation of the venous coats than of a coagulating thrombus in their interior as the primary condition. We find then three elements in the cases: patchiness, metastasis, and symmetry, the latter not always to be met with however. I have mentioned the pain which sometimes attends the onset of the attack. Too little attention has hitherto been paid to the pain which is sometimes present in veins which present no signs of endo-thrombosis. In France attention has been directed to this point, and it pertains, so far as I know to the subjects of a gouty habit of body. It is sometimes so severe as to be mistaken for neuritis, but there is usually some dilatation of veins associated with the pain, and there is no evidence of thrombosis of deeper veins, such as œdema, in the affected area or in the parts below. Plugged veins lying deeply, and of large

size, may not be palpable owing to their depth or the presence of much fat in the integuments, but the evidence in the affected limb is obvious enough, as there is present œdema, with a cool and pallid skin, and sometimes dusky mottling owing to distended superficial veins.

We may now consider the course of gouty phlebitis. In simple and limited attacks involving superficial veins, including veins already varicose, with care and prudence, the pain, as a rule, passes off in a few days, and the inflammatory area of the part also subsides. The cord-like condition is apt to remain for some weeks, but slowly becomes less palpable, disappearing absolutely within a month or six weeks. In graver cases, the thrombus appears to become incorporated with the inner wall of the vein and so to seal up the lumen of the vessel, the circulation being taken on by adjacent veins. Most of us have, happily, an apparent superfluity of veins, and therefore abundance of vicarious channels for the locally impeded circulation. The primary change in these cases is probably, as I have stated, in the inner coat of the vein, and the altered plastic and sticky exudation here leads to stasis and delay in the onward passage of the blood, with the ultimate production of clot or thrombus. We have to regard this process as a veritable localized gouty inflammation, determined by the alighting of specific elements in the toxic blood (uricæmia).

The future course of the attack, whether good or evil, depends actually on the condition of the clot and its subsequent behavior. With appropriate treatment, and prudent management of the patient, we may expect in the majority of cases no untoward results. That such may occur, however, is very certain.

We hope for a gradual removal of the clot, or, failing this, for the sealing up by firm consolidation and fusion of the thrombus and the tunica intima of the vein with a favorable termination.

The untoward result I have mentioned relates to a perverse quality of the thrombus which neither disposes of itself, nor forms adhesions to the venous wall, but remains loose or degenerates, and becomes mobile. In such a case there is always a risk of dislodgment of the clot in whole or in part, and of its being swept along in the upward blood-current into some other part of the body. Now, such mobile thrombi are unfortunately very apt to

be swept up into the vena cava and to lodge in the right auricle and ventricle of the heart whence they are projected rapidly into the pulmonary artery or the lung, with the immediate production of alarming symptoms, faintness, dyspnœa, and giddiness, followed not seldom by death within a few minutes. A necropsy reveals how this has come about. In a less severe case, fragments or plugs may be detached from involved veins, the process having crept up along larger veins, and find their way into the lung, leading to pulmonary apoplexy, with bloody, mucous expectoration. A good many cases have now been noted in which these complicating accidents have occurred, and we have always to bear the possibility of them in mind in any case, however simple it may appear in the first instance. Several of such pulmonary attacks may occur in the course of one of the severer forms of this ailment, and the last may prove fatal. Gouty phlebitis is apt to recur, and it is apt to be one of the lesser and irregular forms of so-called suppressed or abarticular gout. There is generally a history of this disease in the family, if not manifested in a frank or classical form in the patient himself, and hence the true nature of the ailment may not be realized by the patient or his attendant.

The *treatment* of this ailment is simple. Rest is essential for any part in which it occurs, and as the lower limbs are most frequently the seat of it, these should be raised and placed in an easy position. The most favorable one is that with the limb slightly flexed at the knee, with the foot everted. A long pillow may be laid underneath, raising the foot and supporting the hamstring muscles. A piece of lint, doubled, soaked in a lotion consisting of one part of belladonna liniment and three of hot water is then applied over the affected part and covered over with oil silk and a light bandage. Two grains of calomel, with eight grains of colocynth and henbane mass, should be given over night, and an ounce and a half of compound senna mixture with half a drachm of wine of colchicum should follow this early next morning. The diet should be plain, and consist of weak tea and farinaceous food, with white fish and scrambled egg. Light puddings and well-cooked green vegetables may be added. Milk is to be avoided as tending to promote hypermicosis or coagulation of the blood. Extra water drinking or weak lemonade is advisable, and alcohol in the

form of good whiskey must be given, or withheld, according to the age and circulatory condition generally of the individual. The next medicine may be prescribed in the form of carbonate of ammonia with potassium citrate and a few minims of tincture of nux vomica, and wine of colchicum. A few days of rest usually leads to general amendment, and if no extension of the clotting process occurs, all may go on favorably, the pain and inflammatory condition subsiding. No sort of massage must be permitted. I have known death to result from this at the hands of an ignorant old apothecary, owing to dislodgement of a clot. The time varies in different cases as to the dissipation of the thrombosis. Six weeks sometimes elapse before this occurs satisfactorily, but in slight cases, the limb may be carefully used after three weeks. It it very important to warn the patient against getting into bed in the ordinary way by kneeling on the affected limb. This has caused death in several cases. The proper way is to sit on the side of the bed, and to have the leg slowly lifted up. In severe cases there is apt to remain some fulness of the limb, hardly amounting to œdema, but sufficient to induce a clumsiness. Superficial veins may enlarge when deeper-seated ones have been involved and ultimately sealed up. Later on, some gentle massage may be helpful in restoring the circulation. The diet and habits of the patient require attention so as to keep down any gouty tendency; otherwise, there may be a recurrence of the process, not seldom in the vicinity of the primary attack. A visit to some suitable "spa" is advisable, and all measures which can restore the individual to his highest attainable health should be availed of.

NOTE.—If any of you desire to learn more about gouty phlebitis, I would urge you to read Sir James Paget's original paper on the subject,[2] and an admirable account of some cases by the late Dr. Tuckwell, of Oxford.[3] An excellent "Treatise on the Etiology and Pathogeny of Phlebitis,"[4] by M. le Dr. Ernest Poulain of Bagnoles-de-l'Orne (Prix Mège, 1907), is also well worthy of study.

[2] *St. Barth. Hosp. Reports*, vol. ii, p. 82, 1866.

[3] *Ibid.*, vol. x, p. 22, 1874.

[4] Ouvrage couronné par l'Académie de Médicine, Paris (Asselin et Houzeau, Paris, 1908).

THE DUTY OF THE PHYSICIAN IN THE PREVENTION OF ORGANIC DISEASE OF THE HEART *

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GENTLEMEN: This patient (Case I), a male, aged thirty years, comes to us with the history of rheumatism. My principal object, however, in bringing him before you this morning is not to speak particularly in reference to rheumatism, but of the duty of the physician in preventing attacks of acute rheumatism from resulting in serious structural disease of the heart.

I do not know whether or not this man has heart disease, as I have not yet examined him. He was brought here so that we might examine him carefully with a view of determining whether there is any organic lesion of the heart. He says that he first had rheumatism six months ago, that he was confined to bed for a week or two and that he suffered considerable pain about his knees, in both arms, and also in his neck.

Now, in the course of development of organic lesions of the heart, we know that valvular disease (valvulitis) is generally due to, or follows some infective process, most frequently rheumatic fever, and if we see such a patient in the beginning of the attack of acute rheumatism I believe it is possible, if we receive proper assistance from the patient, to prevent the occurrence of heart lesions which may remain throughout the balance of life, resulting not only in serious impairment to health, but perhaps rendering the patient an invalid the rest of his life.

Of all the various valvular lesions of the heart, the mitral valve is the one most frequently affected. As is well known the first sound of the heart is produced in consequence of two factors, viz.: muscular contraction of the heart, and closure of its valves. It is the duty of the physician when he is in charge of a patient

* Clinical Lecture delivered at the University of Louisville, Medical Department.

who has an attack of acute rheumatism to watch carefully the condition of the patient's heart, and be on the alert for any modification of the sounds. This necessitates frequent and repeated examinations of the heart during convalescence and for some time thereafter.

This man says that he has had rheumatism and it is probable that his statement is true. Now one reason that physicians have a great deal of trouble in the treatment of heart disease, is that they do not receive proper assistance from the patient. He does not realize the necessity of obeying the injunctions of the physician in reference to taking care of himself under the circumstances; he wants to get out of bed as quickly as possible, and does frequently get out of bed and leave the room before he should. This results in serious and indeed often permanent injury to himself. As we supposed, this man says that he got out of bed and went back to work just as soon as he could.

When we are called upon to treat a case of acute inflammatory rheumatism we generally allay the pain by the use of adequate doses of some salicyl compound, the administration of morphine, enforced rest and the use of various applications to the inflamed joints; and after a course extending over a period of two or three weeks, perhaps more, our patient emerges from this attack of acute inflammatory rheumatism more or less weakened and anæmic, perhaps with some beginning changes in the valve structures of his heart, to which we have not given sufficient attention, and to which we have not listened with sufficient care.

If there is a time when good can be accomplished by treatment of valvular disease of the heart, it is when the valves first become the seat of inflammatory changes and not after these alterations have taken place and the vegetations of disease processes affecting the valves have become permanent. I think we are oftentimes negligent in the treatment of this class of patients. When, in the course of an acute attack of inflammatory rheumatism, there is evidence of threatened or beginning involvement of one or more valves of the heart we do not insist sufficiently upon the proper amount of rest in the recumbent position, and if necessary enforced rest in bed.

We must bear in mind the two particular structures which are

usually involved in rheumatic affections, the tissues of the joints, and the endocardium, and one of the most important indications for the cure of either one is rest. For the former we can secure more or less permanent rest by having the patient remain quietly in bed, but the heart is an organ which of course it is quite impossible for us to put at entire rest. Yet by insisting upon the patient remaining quiet for a sufficient interval during and after recovery from an attack of acute inflammatory rheumatism, especially where there are indications that there may be a beginning lesion of the heart, and by the administration of appropriate remedial agents we can often prevent the development of serious valvular disease in after life; and if valvular change has already taken place, we can by proper medication and rest modify and perhaps bring about permanent cure before the condition has progressed to the point where there is permanent stenosis or regurgitation of one or more valves of the heart.

Case II.—The next patient, a male, aged forty-two years, applies for treatment with the diagnosis already made of "heart disease." Of course to those of us who are familiar with diseases of the chest this diagnosis, even if it be correct, means very little. From our physical examination of the patient we have to determine in just what the disease of the heart consists. In this connection, I have in mind the case of a boy who was recently run into by an automobile. Claim had been made by the parents and attending physician that as a result of the accident the boy suffered permanent injury of the heart. I examined the boy carefully yesterday, and failed to discover any indication of heart disease! It is not my purpose, however, at this time to take up the consideration of the various diseases which we may see in this clinic, except as they come before us.

The diagnosis of heart disease, like the diagnosis of lesions involving the lung, often means the careful exercise of all the faculties with which we are possessed. And I desire to remark, in passing, as I have said before, and I speak as a laboratory man, that the tendency of modern times has been to depend entirely too much on laboratory methods to the exclusion of clinical investigation. We have been taught that whenever tubercle bacilli are found in the sputum that this enables us to make an early diagnosis

of pulmonary tuberculosis. I have seen many cases of incipient tuberculosis in which there were absolutely no tubercle bacilli discovered in the sputum! We must bear in mind that the tubercle bacilli are not ordinarily found in the sputum until the disease has reached the point where there is destruction of lung tissue, it may be the third stage, where cavities have formed from the breaking down of the caseous material and discharge of these masses into the bronchi, and their subsequent expectoration. Of course our ability to find the tubercle bacilli depends entirely upon their presence in the sputum, and it is perfectly possible in the early stages of pulmonary tuberculosis, when the diagnosis is most important, if we rely solely upon laboratory methods, we may be utterly unable to make the diagnosis. There may have occurred no breaking down of lung tissue, no discharge of tubercle bacilli, but physical examination of the patient's chest will always enable us, even in the early stages of the disease, in the majority of instances, to make a correct diagnosis, particularly if we consider carefully the history of the case. When we have established the diagnosis by clinical means, we will find our conclusions will be confirmed more often than not by laboratory methods.

I do not mean to underestimate laboratory diagnosis because it occupies an important place in medical investigation, but what I say is,—without fear of contradiction, because I think I have had sufficient personal experience to know,—that we are not justified in turning over specimens of sputum, urine, blood and other secretions to the laboratory where the work is done usually by inexperienced men, and expect them to make a diagnosis which we as clinicians are unable to do. I must insist that the clinical study which we take up here is of much more value to the advanced student and the average doctor than the oftentimes useless examinations of sputum for tubercle bacilli when the organisms are present in the lung but are not being discharged!

When we ascertain from the clinical history and physical examination that there are conditions in the patient's chest to warrant the suspicion that abnormal changes have taken place in the structures of the lung, then it is that we may put the patient under such treatments and surroundings as will result in benefit, long before he has reached the point where the tubercle bacilli make

their appearance in the sputum. On the other hand, there is a class of cases with which we come in contact where the diagnosis of typhoid fever instead of phthisis pulmonalis has been made, and the patient treated for a considerable time on this basis. There may be early breaking down of the lung tissues, a rapidly progressing tuberculous peribronchitis, and caseous pneumonia with rapid destruction and discharge of detritus with the sputum. Under these circumstances of course the laboratory method should take precedence over clinical signs. Unless I know something about the history of the case, the clinical symptoms, and if possible the condition of the patient, as a laboratory man I simply state what I find and ask permission of the clinician to place my report alongside of his clinical findings, and thus make his own diagnosis.

From your studies of the physiology of the heart, you will recall there are normally two sounds in connection with its action, known as the first and second sounds of the heart. The first is a rather prolonged sound and may be phonetically represented by the word *Lubb,* the second is a much shorter and sharper sound and may be phonetically represented by the word, *Dupp.* In making an examination of the chest, especially for suspected disease of the heart, always strip the patient to the waist. It is better in making your examination, however, to have some slight fabric like a piece of muslin, woolen, cotton, even a flannel undershirt, or handkerchief, between your ear and the chest. You must bear in mind that in some individuals the chest is quite hairy, and these hairs scraping against the ear may produce sounds which in many respects resemble crepitant râles. However, we should not neglect the use of the stethoscope in learning to recognize abnormal sounds of the heart and lungs. We may be able to hear them with the normal ear, but they are magnified and of course heard more distinctly with the stethoscope. Always have the patient stand facing the light, and the source of light so arranged as to fall equally on the two sides, so that it does not cast a shadow on either side of the chest.

We have carefully examined the patient before us, and my impression is that there are three distinct murmurs in this case: (1) aortic stenotic, (2) aortic regurgitant, and (3) mitral regurgitant. Though valvular disease may be induced by any in-

fectious process, such as scarlet fever, diphtheria, typhoid and typhus fevers, possibly pneumonia, the largest percentage (I believe more than forty-seven per cent.) of cases of valvulitis are the result of rheumatic affections. The patient before us has not had rheumatism so far as we can judge from his history, but he has been the subject of another disease which is often the cause of atheromatous changes in the blood-vessels,—syphilis. It may be on further investigation that we will find an arteriosclerosis which has been induced by the specific disease which he has had. Such cases are not uncommon.

As you know the heart is nothing more nor less than the pump which maintains and hastens the onward flow of blood through the circulation. If as a result of disease the valves of the heart become clogged, or disorganized, if as a result of inflammatory exudates there is a deposition of scales (calcareous deposits) upon the valves, then of course they do not properly fit, they may stand out so to speak in the blood stream, there is an impingement upon the channel through which the blood must pass. In consequence of the stiffened or ill-fitting valve naturally the orifice is considerably lessened in size, and in order to maintain the same amount of blood through the circulation the heart must beat with much greater force. In endeavoring to force the required amount of blood through this narrow orifice, the heart undergoes a condition of compensatory hypertrophy, and when this compensation becomes thoroughly established the condition of the patient may be as good apparently as it ever was so long as it is not subjected to over-strain.

Then, again, as a result of some inflammatory process one or more segments of the valves may become bound down by inflammatory adhesions, and in consequence of this, if the mitral valve is diseased when the ventricle contracts, the left auriculoventricular opening is not perfectly closed and the blood is driven out in two directions, some going out into the aorta as it should and some going back into the auricle; thus the heart has to work with greater force and more strain is placed upon it in order to overcome the deficiency which is consequent to the diseased process set up in the mitral valve.

As stated before, at each of the four sets of valves there may

occur two conditions which will produce abnormal sounds: (1) insufficiency, and (2) stenosis. One of the best descriptions of the changes in sounds produced by stenosis of the valves of the heart that I have ever read was in Tanner's "Practice of Medicine," in which he compared the current of blood flowing between the cavities of the heart to a large placid stream of water like the Ohio River which flows along perfectly quietly without sound or ripple; the moment you build a bridge across that river, putting piers in some of the space occupied by the original bed of the river, you narrow the channel and in consequence the water which before was passing along quietly without sound, now as a result of narrowing of the channel becomes a rapidly flowing stream which gives rise to considerable noise as it rushes through the narrowed orifice! We have exactly the same condition when there is a stiffened valve standing out in one of the openings of the heart, be it the aortic, the pulmonary, the mitral, or the tricuspid opening, and in consequence of the obstruction we usually have a hissing sound replacing or magnifying the natural sound, generally replacing it but not invariably so.

While of course any and all the sets of valves of the heart may be affected with primary valvulitis, I desire to say that the condition is much more frequent on the left side, that inflammatory affections of the valves on the right side are usually secondary to lesions of the left side. Therefore, from a practical standpoint, I believe it to be a good rule in examining a patient with suspected heart disease first to determine or satisfy ourselves that the abnormal sound is upon the left side, although in the beginning we may assume this to have been true almost to a certainty; let us then determine if the murmur accompanies the first or the second sound of the heart. A good working rule is that if the murmur accompanies or replaces the first sound of the heart and is heard plainest at the fifth intercostal space on the left side, it is generally one of two murmurs, *i.e.*, either a mitral regurgitant, or an aortic stenotic. If the murmur accompanies or replaces the second sound of the heart and is heard plainest at the second intercostal space on the right side, it is usually a murmur resulting from aortic regurgitation. Having this starting point firmly fixed in our minds, if we can determine from that where the lesions are

situated, then subsequent differentiation of other lesions of the heart is not difficult, and the diagnosis is really a comparatively easy matter.

I have said nothing about mitral stenosis in this connection. A murmur produced by mitral stenosis as a rule is heard plainest in the so-called mitral area, and unlike the murmur of mitral regurgitation, is transmitted to the right, the murmur of mitral regurgitation being transmitted to the left and frequently heard up under the scapula.

In the case before us we can hear a sound which is evidently a murmur, the result of mitral regurgitation as it is transmitted backward and to the left. Then upon listening above we can hear a hissing sound which entirely replaces the second sound of the heart. The first sound of the heart, as we know, is a combined phenomenon produced by two elements, one of which is a contraction of the muscular walls of the heart, the other a flapping of the valves accompanied by the rush of blood through the various chambers. The second sound of the heart is purely a valvular sound, and is produced solely by a closure of the semilunar valves guarding the aorta and the pulmonary orifices.

In the normal circulation through the heart we must bear in mind that the blood is brought from the lungs to the left auricle and remains there until the auricle becomes slightly dilated. The same phenomenon is taking place in the right auricle. When these two chambers have become filled with blood, muscular contraction takes place which forces all the blood they contain down into the ventricles, which in their turn are thrown into a condition of over-distention. Then follows a slight pause, contraction of the ventricles then occurs and the blood is driven out from the left side of the heart into the systemic circulation, and from the right side to the pulmonary circulation. So long as the valve structures remain intact these phenomena recur uninterruptedly. The blood is returned to the chambers of the heart through the systemic circulation, back through the superior vena cava to the right auricle, then to the right ventricle, to the lungs where the blood is aerated, then back to the heart and again to the systemic circulation. This process is carried on continually with but slight intervals of rest for the heart.

The heart is an organ which is never completely at rest, although there is a slight period of rest between beats. It is unlike other organs of the body in this respect; consequently the heart is more likely to be permanently damaged than are the joint structures of the body following an attack of acute inflammatory disease, since the joints and other organs of the body can generally be placed in a condition of complete rest.

In every case of heart disease we should take into consideration the period during which the heart is occupied by actual work. Of course the heart possesses a certain amount of reserve force which can be called into play on extra-exertion. Those of us, for instance, who have normal hearts, when called upon to do so, could run a mile or two without any serious inconvenience; but ask an individual suffering from heart disease to run a mile and he would not go a great distance before the heart would begin to show evidence of distress from over-work in its effort to maintain the circulation during the increased muscular exertion. The natural reserve force of such an individual has become impaired, even during the period of so-called rest his heart is working in an effort to maintain normal circulation, and when he endeavors to call upon it for the reserve force there is little response; in fact in serious advanced heart disease all the normal reserve force is gone. That is one reason why in the treatment of acute rheumatism I always insist upon early and prolonged rest in the hope of preventing subsequent permanent damage to the valves of the heart, because a valve once diseased usually remains so. While there are cases where heart murmurs undoubtedly due to valvulitis have disappeared, yet where the damage is extensive as a rule it can never be repaired. The best we can do under such circumstances is to train the patient to the idea that he must live a life that is different from that of other and normal individuals.

In a case of pure aortic regurgitation, what is the condition of things present? The mitral valve may be intact, or it may present a condition of insufficiency as a result of dilatation of that orifice. Some of the segments of the semilunar valves may be bound down or shrunken so that the aortic orifice is no longer properly closed, or the edges of the valves may be the seat of vegetations which prevent proper closure. What follows when the

heart contracts? At the beginning of cardiac systole we have first contraction of the auricles, followed by a short pause, then contraction of the ventricles. If the semilunar valves close properly the blood is now driven out into the aorta, thence through the general circulation. If we recall our anatomy we will remember that the large sized arteries are possessed of a coat which contains considerable tissue that is exceedingly elastic. When the ventricle is thrown into a condition of over-distention by becoming filled with blood and contraction occurs, naturally the valve ought to close the moment ventricular contraction ceases; but if the valve cannot properly close because of its being bound down by adhesions, perhaps only one segment falling into place, what happens? Because of failure of the valve to close there is nothing to prevent the blood from flowing backward into the ventricle, and that is exactly what happens. The aorta being elastic closes down, so to speak, on the blood and forces most of it forward in the direction of the general circulation, but some of it goes backward in the direction of the ventricle from which it came, and the result is considerable interference with the circulation. Many of you have probably noted the phenomenon known as Corrigan's, or the so-called water-hammer pulse, in which the artery alternately becomes fully distended then suddenly collapses. The volume of the pulse is large, always imparting considerable movement to the radial artery. This character of pulse is always suggestive of aortic regurgitation.

As a natural consequence of aortic regurgitation, in order to overcome the deficiency, the heart has to take on greater action, its muscular fibres undergo increased development and it becomes the subject of hypertrophic enlargement. The left ventricle participating in the hypertrophy may undergo degeneration, and as a result of that condition the mitral orifice may become involved; there may be no disease of the valve segment, but the mitral orifice may become decidedly enlarged; the valve no longer closes the enlarged orifice, consequently we have at that point what is known as mitral insufficiency, brought about by disease primarily affecting the aortic valve, a condition of relative mitral insufficiency. When this becomes manifest, or possibly even before, in consequence of the damming back of the blood upon that side of

the heart, we have dilatation of the cavity; and the dilatation in those cases of pure aortic regurgitation is sometimes simply enormous, giving rise to the condition known as cor-bovinum or ox-heart. Associated with that condition there may be, as I think there is in the case before us, a condition of stenosis of the valve, where the valve is stiffened and unable to close and not only presents an obstacle to the outward flow of blood but allows it to come back through imperfect closure of the valve.

In all forms of heart disease, if we are called upon to express an opinion as to the danger to life dependent upon the development of these valvular lesions, I believe we can safely say that mitral regurgitation is the most favorable, aortic stenosis second, mitral stenosis third, with aortic regurgitation last.

Surgery

REPORT OF FIFTEEN CASES OPERATED UPON IN PROF. W. L. RODMAN'S SURGICAL CLINIC AT THE MEDICO-CHIRURGICAL HOSPITAL

BY J. STEWART RODMAN, M.D.
PHILADELPHIA, PA.

OMENTAL (?) CYST

Case I.—A. R., female, white, 17 years of age. Patient comes to hospital complaining of swelling of abdomen.

Family History.—Negative.

Past Medical History.—Except for the usual diseases of childhood has been very healthy. Puberty at fourteen. Menses regular and painless.

Present Illness.—In 1905 patient states that for first time she noticed that her abdomen was slightly enlarged. She insists that swelling began from above downward. Abdomen continued to increase in size. Suffered no pain and had no shortness of breath. There has been no œdema of feet or ankles, and has never been forced to arise at night to void urine. In March, 1907, entered the medical ward of the Medico-Chirurgical Hospital under the care of Prof. Judson Daland. She remained in the hospital for two months and during this period her abdomen was tapped twice. At each tapping a large quantity of clear colorless fluid was obtained. Patient was discharged from the hospital June, 1907, after second tapping. Four months later swelling reappeared and has steadily increased until present time. During the past month she has been very uncomfortable at times, short of breath and has been often forced to arise once or twice during the night to void urine. There has been no œdema of feet or ankles.

Physical Examination.—A rather pale, slightly emaciated girl; tongue slightly coated, thyroid gland moderately enlarged. There

is no enlargement of the superficial lymph-glands. Chest: Lungs are clear throughout. Heart: Apex beat neither visible nor palpable; outline of cardiac dulness normal; heart-sound clear. There are no murmurs but there is an occasional reduplication of the second sound. Abdomen: Very much enlarged, measuring 41 inches at the umbilicus. Liver dulness from the fourth interspace to costal margin. The enlargement of the abdomen is general, no tumor mass is palpable, and there is marked movable dulness in flanks and a good transmission wave on percussing either flank.

Bimanual Pelvic Examination.—Shows œdema of the vulva, normal cervix though rather low, considerable bulging of the anterior and posterior vault of the vagina.

Urine—negative. Blood—hæmoglobin 85 per cent.; erythrocytes, 4,125,000; leucocytes 9000.

On October 31, 1908, abdomen tapped for the third time and thirteen litres of a clear colorless fluid removed. Examination of this fluid shows sp. gr. 1007, colorless, faintly acid, no albumin, sugar, or bile pigments; large amount of chlorides; microscopically no formed elements.

Clinical Diagnosis.—Intra-abdominal cyst, probably of pelvic origin.

Operation, November 14, 1908.—Ether anæsthesia, median incision from umbilicus to symphysis pubis. On opening the parietal peritoneum it was seen that the entire peritoneal cavity was filled up by an enormous unilocular cyst. The cyst was partly evacuated by means of a trochar and canula. The cyst wall stripped easily from the parietal and visceral peritoneum and the pedicle was soon found to have attachment in the upper abdomen. The intestines were pushed upwards and backward by the cyst being wholly posterior to it, and the overhanging layer of omentum extended half way down over the anterior surface of the cyst wall. The entire cyst was finally stripped free from the posterior surface of the great omentum and removed from the abdominal cavity. Glass tube drainage was inserted to the pelvis and the wound closed by through-and-through sutures of silkworm gut. The patient's condition was good on leaving the operating table. The drainage was removed at the end of forty-eight hours and except for a fast and rather irregular pulse during the first three days of convales-

Fig. 1.

Omental Cyst.

Fig. 2.

Omental Cyst.

cence the patient made a splendid recovery and reported herself well one week ago—a year following the operation.

Pathological Report.—A very large unilocular cyst; microscopical examination of cyst wall shows only fibrous connective tissue; no epithelium (Figs. 1 and 2).

SARCOMA OF BRAIN

Case II.—C. D., male, white, 25 years old, single.

Chief Complaint.—Patient comes to hospital seeking relief from epileptic convulsions which begin on right side of body, in fingers of right hand, and spread to right arm, leg and right side of face.

Family History.—Negative save for fact that one sister had chorea.

Previous Medical and Social History.—Has had usual diseases of childhood. No serious illness other than present affliction. Does not use alcohol, but does use tobacco moderately. Denies specific infection.

Present Illness.—About three years ago convulsions first began with twitching of fingers of right hand, followed by slight clonic contractions of right arm. Patient states that he has absolutely no memory of traumatism to head, and states that he is at a loss to account for these seizures. Twitching of right hand, arm, right leg and right side of face became progressively worse, though at first did not lose consciousness during these attacks which would occur during first year and half at intervals of about one month. Later began to lose consciousness during attacks and felt a vague forewarning of the seizures. At this time one year ago, patient was admitted to the surgical ward of another Philadelphia hospital, where he shortly afterward was operated upon. For six months after operation he remained apparently much improved, having had only two or three mild epileptiform attacks, without losing consciousness. During last year, however, and especially during last six months, seizures have been severe and frequent, he having had three or four attacks in a single week. He states that attacks continue to start with twitching of fingers of right hand, and relatives say that it is the right side of the body which shows the more severe convulsive movements during hard attacks.

Physical Examination.—Patient is a fairly well developed young man. His expression is somewhat dull and listless, right eyelid drooping slightly, and right corner of mouth noticeably drawn down. Pupils react to light and accommodation. Skin slightly pale. Superficial lymph-nodes show no evidence of enlargement. Examination of left side of head shows a depression in left temporal region, and scalp shows " U " shaped scar, evidently due to the healing of a flap reflected at previous operation. Lungs appear clear throughout, and heart is normal.

Patient says that his right arm is weaker than left. Examination failed to reveal any paralysis of this arm, however, although it does not seem to be as well developed as its fellow. Patellar reflexes are seemingly normal, as are elbow-jerks, etc. There is no ankle clonus and no Babinski sign present.

Urine — negative. Blood — leucocytes 8000; erythrocytes 4,300,000; hæmoglobin 85 per cent. (Dare).

The patient and his family were so anxious that he should be given the small chance of benefit that an operation held out to him, that it was finally decided to perform a craniotomy. This decision was rather reluctantly arrived at, however, and not until patient had been under the careful observation of the Neurologist, Dr. T. H. Wiesenburg, who assured us that the epilepsy was undoubtedly of a Jacksonian type, and probably, in his judgment, due to a lesion involving the left motor area.

Operation, April 2, 1909 (Dr. Burns).—Under ether anæsthesia and with patient in semi-sitting posture, a " U " shaped incision was made in left temporal region. On carrying the incision through temporal muscle to the bone a depression was seen, evidently the result of a previous osteoplastic craniotomy. The bone was cut through following the old line of healing, and an osteoplastic flap turned back. The dura now immediately bulged forth into the wound, and appeared to be under tension. A curved incision reflected a flap of dura. At this point, considerable difficulty was experienced in checking hemorrhage, as several small veins had been cut across in opening the dura. As soon as the field was cleared from hemorrhage a distinct bulging of the brain substance was seen, reddish gray in color, over the surface of which ran many blood-vessels. Evidently there was a tumor mass involving this area, but in

attempting to explore further, more hemorrhage was encountered. Patient's condition became alarming and all further attempts at exploration abandoned. It did not seem justifiable to remove any brain tissue for pathological examination. Dr. Wiesenburg, who was present at the operation, expressed the opinion that the tumor was probably sarcoma. The dura was partially closed, a small gauge drain being inserted, as it was impossible to check all hemorrhage. Osteoplastic flap replaced, temporal muscle sutured, and incision closed with drainage. External pressure was applied by means of a tight bandage. Patient had rallied somewhat on leaving table—pulse 120, fair volume. When seen in the ward shortly after operation condition seemed fairly good, temperature 96.8°, pulse 120, respiration 20. Had not recovered from anæsthesia. About one hour later complained of pain in head, and had several convulsions in rapid succession. As these continued following one another so rapidly as to practically induce a condition of status epilepticus, patient was again taken to operating room, wound reopened and large subdural clot found and removed. His condition improved somewhat temporarily, but convulsions reappeared and patient rapidly succumbed, dying about eight hours after operation.

ANGIOMATA OF FACE

Case III.—J. E. B., female, white, four months.

Family History.—Negative.

Previous History.—First child, normal delivery, has been breast fed and healthy since birth.

Present Illness.—At birth small, so called, "birth marks" noticed on both sides of face, both being in front of ears. These were small, bluish swellings at that time, and have steadily increased in size since then. Tumor of left side of face has grown more rapidly than that of opposite side, and within last month mother noticed that lower lip was swelling.

Physical Examination.—A large tumor is seen in front of either ear, the left, about size of hen's egg, the right somewhat smaller. These tumors are bluish red in color, rather soft, and seem to be typical cavernous angiomata. The lower lip is swollen and is also the seat of an angioma. Otherwise, the baby seems normal and well developed (see Fig. 3).

Treatment.—Tumor of left side injected with hot water, passing needle through mouth October 6, 1908, by Prof. Wyeth, of New York, who was invited to hold Dr. Rodman's clinic to demonstrate his method of treating angiomata. Tumor of right side injected October 8, 1908. Injections were carried out at intervals of three to four days for a month after admission to hospital. Save for a hardening of tumor of right side of face, there was no improvement in condition, there being apparently no attempt at coagulation on left side. As further treatment seemed inadvisable, patient was discharged.

SQUAMOUS CARCINOMA OF LIP

Case IV.—W. T., male, 51 years old.

Chief Complaint.—Patient comes to hospital complaining of "sore" of lower lip.

Family History.—Father died at 65 of what was diagnosed as cancer of liver. Except for this, family history negative.

Previous Medical History.—Always healthy. Remembers no severe illness.

Present Illness.—Patient has always been a great smoker, especially smoking a pipe. Four months ago a small ulcer appeared on left side of lower lip. This has refused to heal since then, and occasionally discharged a bloody pus. Has not been painful. On consulting physician, was advised to have this ulcer treated surgically.

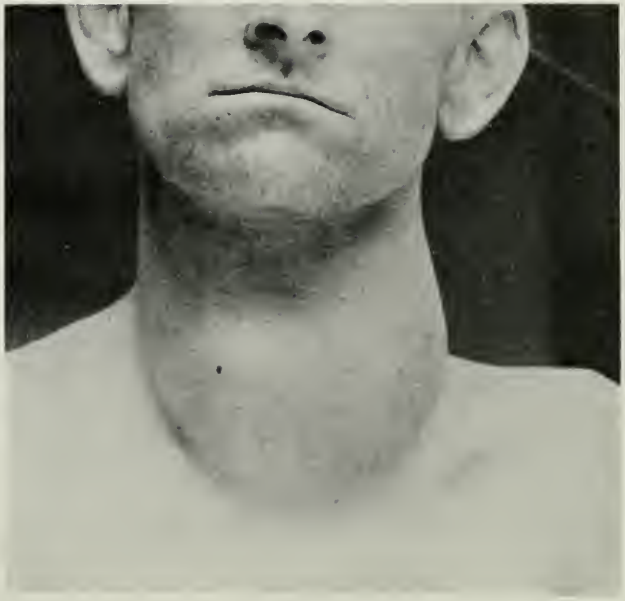
Physical Examination.—Well developed man. General examination of lungs, heart and abdomen failed to reveal any abnormality. Examination of lower lip shows an ulcer with irregular edges, is not indurated. Submaxillary and submental glands are not enlarged to palpation.

Operation, February 9, 1909.—Ether anæsthesia. Following our usual plan of procedure, a transverse incision was made across neck above upper border of hyoid bone. By dissection the submaxillary, salivary, lymphatic glands, and submental glands of both sides of neck, were removed "en bloc." This we do in all cases of carcinoma of the lower lip, believing that it is safe to assume that such glands may be contaminated in these cases, despite macroscopical evidence to the contrary. After finishing neck dissection and closing wound with horsehair sutures, a large

FIG. 3.

Congenital angioma of face.

Fig. 4.



Unilateral cystic goitre.

Fig. 5.

Unilateral cystic goitre.

"V-shaped" portion of the lower lip embracing the ulcer and a considerable portion of tissue on either side of it was removed by incision, and the edges of the wound cauterized by thermocautery. Edges approximated with silkworm gut. Patient had a speedy and uneventful recovery.

Pathological Diagnosis.—Squamous cell carcinoma of lower lip. Submaxillary and submental glands normal.

UNILATERAL CYSTIC GOITRE

Case V.—H. L. W., male, white, 41 years old.

Chief Complaint.—Comes to hospital March 13, 1909, complaining of tumor of neck.

Family History.—Three brothers and two sisters, none of whom have had goitres; in fact family history is entirely negative.

Previous Medical History.—Pneumonia about ten years ago.

Present Illness.—Seven years ago first noticed swelling of neck. This began on left side and at that time applied iodine, which seemed to reduce swelling. It soon returned, however, and tumor has gradually increased in size until present time. Patient states that he experienced absolutely no discomfort until two months ago, when he first noticed slight difficulty in breathing, and began to cough. Dyspnœa has increased since then, until now it requires almost constant effort to breathe. The cough has become somewhat hoarser and during last few weeks has had some pain in region of tumor. At no time have his eyes been prominent, nor have there been tremors or gastric crises. Has never had palpitation.

Physical Examination.—Patient is rather emaciated and presents prominent tumor of neck (Figs. 4 and 5). Breathes with difficulty. Skin and mucous membranes are somewhat pale. Eyes show no exophthalmos and pupils react to light and accommodation. Examination of neck shows very prominent tumor involving left side, pushing thyroid cartilage to right. In fact so far has the tumor encroached upon the right side that it seems to fill up neck, especially from anterior view. On palpation the tumor at first was taken to be solid, it was so resistant, but more careful examination revealed a point of slight fluctuation, indicating, perhaps, fluid under pressure.

Examination of lungs proved them clear throughout; outline

of cardiac dulness normal; apex beat in fifth interspace in mid clavicular line; heart sounds are distinct, no murmurs; abdomen negative; there is no enlargement of superficial lymph-glands; pulse (radial) regular, good volume, sixty per minute; urine—negative.

On the day following admission to the hospital, March 20, 1909, tumor was tapped and about 350 c.c. of bloody grumous fluid removed. Dyspnœa at once became less marked (Fig. 6).

Operation, March 23, 1909.—Chloroform anæsthesia. Transverse "collar" incision, entire left lobe of thyroid gland found to be cystic, and to have pushed thyroid cartilage and trachea far to right side. Trachea was found to be also compressed. Slight difficulty was experienced in separating cyst from trachea, but patient's condition remained satisfactory. Entire cystic left lobe was removed, middle and right lobes appearing normal, being undisturbed. Wound closed and cavity drained with a cigarette gauze drain.

Patient made a rapid and uneventful recovery, wound healing by first intention (Fig. 7).

Pathological Report.—Cystic degeneration left lobe thyroid gland.

ADENOFIBROMA OF BREAST

Case VI.—M. C., female, 26 years old, white.

Chief Complaint.—Comes to hospital complaining of tumor of breast.

Family History.—Negative.

Previous Medical History.—Three years ago was operated upon in this clinic, a small fibroma being removed from right breast at that time. Warren's plastic resection was practised, resulting scar being scarcely noticeable.

Present Illness.—For past two years has had pain at intervals in left breast, but had not noticed any tumor until examined by Dr. W. L. Rodman, March 29, 1909, when a small, rounded, apparently encapsulated, painful tumor of left breast, in lower outer quadrant was discovered. Patient was advised to have this small tumor removed, and accordingly was admitted to the Medico-Chirurgical Hospital, March 30, 1909.

Physical Examination.—Patient is a well developed woman; skin clear; superficial glands are not palpable. Chest—examina-

Fig. 6.

Semilateral cystic goitre showing site of appenetration.

Fig. 7.

Unilateral cystic goitre—final result.

tion of heart and lungs proved entirely negative. Abdomen—negative. Examination of left breast shows a small hard tumor, distinctly encapsulated, painful to pressure; skin seems freely moveable over tumor. Urine negative.

Operation, April 4, 1909.—As tumor was clinically benign, a plastic resection, or J. Collins Warren's operation, was performed. Accordingly incision was made along lower border of breast and entire gland turned back. A small round encapsulated tumor was found and removed. Macroscopic section showed that tissue was apparently fibroma, and this was confirmed some ten minutes later by frozen section report. Wound in breast tissue closed with catgut, and external wound with horsehair sutures—convalescence uneventful.

We cite this case to lay stress upon Warren's highly satisfactory operation. This procedure is one that we have used since first advocated by Warren, to the exclusion of all others. It insures a perfect cosmetic result. The scar is hidden by the overhanging breast itself, and the tissue removed is not missed and does not leave an unsightly depression. Of course, this result is very desirable in young unmarried women, they being the ones most often afflicted with growths of the benign type. The accompanying photograph (Fig. 8) well illustrates how perfect is the cosmetic result.

CARCINOMA OF BREAST

Case VII.—H. W., female, 41 years old, married.

Chief Complaint.—Patient comes to hospital complaining of "lump in left breast."

Family History.—Mother died of "old age." Father had tumor of liver, probably cancer. Three brothers and two sisters living, and well.

Previous Medical History.—Has always been healthy. Has had one child, who is now 7 years old. Menses regular and painless.

Present Illness.—Patient first noticed tumor of left breast four months ago. Does not remember any traumatism to breast and has never had an abscess. Since first noticed, lump has increased rapidly in size, especially during past few weeks. There is no discharge at present from nipple of this breast, but states that about one year ago there was a slight bloody discharge which continued,

however, for but a short time. Has been entirely free from pain and does not think she has lost any weight.

Physical Examination.—Well developed, well nourished woman. Superficial lymph-nodes are not palpably enlarged. Skin clear. Lungs are clear throughout. Heart-sounds clear, no murmurs. Left breast—examination of left breast shows a tumor mass centrally located about size of lemon; tumor is hard, adherent to skin, which is dimpled over growth; retraction of nipple; auxillary glands are not palpable. Right breast apparently normal. Abdomen is negative. Urine—negative.

Clinical Diagnosis.—Carcinoma of left breast (Figs. 9 and 10).

Operation, April 15, 1909.—Ether anæsthesia. In this case the same general plan of procedure was followed as in all of our malignant cases for the past three years. The technic described in detail may be found in Dr. W. L. Rodman's monograph on Diseases of the Breast. The principles underlying the technic may be briefly stated as follows: First the axilla is attacked at the beginning of the operation for three reasons, namely, to avoid, as far as possible, expressing cancer cells along the lymphatic channels, as is done by first manipulating the breast itself; to attack vessels at their root, lessening hemorrhage; and to ascertain whether that particular case is operable or not, judging from the standpoint of lymphatic involvement. After the completion of the axillary dissection, the breast is removed by an eliptical incision, removing the greater and lesser pectoral muscles, and the sheath from the upper half of the rectus muscles. The remaining skin border is freely undermined, which will usually insure easy approximation of the wound edges. Skin grafting is infrequently done, secondarily. The mobility of the arm is not interfered with by a contracting cicatrix, as the incision does not extend on the arm, the axillary dissection being carried out through a straight incision two fingers breadth from the anterior axillary fold.

Pathological Report.—Carcinoma simplex of breast.

PERITYPHILITIS—PERFORATED CÆCUM—ACUTE APPENDICITIS

Case VIII.—A. R., aged 17, schoolgirl.

Chief Complaint.—Pain in abdomen, nausea and vomiting.

Family History.—Negative.

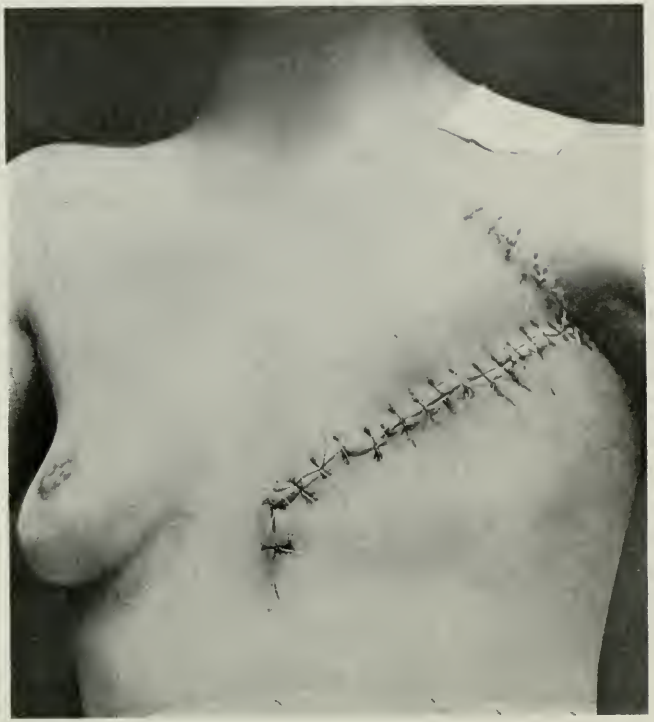
FIG. 5

Adenofibroma of breast showing operative result.

Fig. 9.

Carcinoma of breast.

Fig. 10.



Carcinoma of breast—operative result.

Past Medical History.—Had an attack of pain in right side of abdomen, nausea and vomiting, June 1908—in bed three days at that time.

Present Illness.—Began at 3 A.M., October 20, 1908, with a sudden, violent, colicky pain in abdomen, which was general at first but soon localized to right iliac fossa. Began vomiting almost simultaneously with advent of pain, and continued to do so for several hours. Vomiting stopped after twelve hours, but pain became more severe and continued until admission to hospital, thirty-six hours after onset of symptoms. Patient says that she had eaten "veal potpie" for supper on night of illness. Bowels have always been regular.

Physical Examination.—Patient found lying on her back in bed evidently suffering intense pain—knees drawn up—cheeks are flushed, tongue thickly coated. Temperature 102 degrees, pulse 120—small, weak and slightly irregular.

Examination of lungs and heart showed nothing abnormal. Abdomen slightly distended, exquisitely tender to palpation, especially over right iliac fossa. Both recti were very rigid, especially right rectus. Blood examination—leucocytes 24,000. Urine—negative.

Clinical Diagnosis.—Acute fulminating perforative (?) appendicitis.

Operation, October 21, 1908.—Patient was operated upon immediately after admission to hospital—ether anæsthesia. Incision through external and internal oblique muscles well over toward crest of ilium. On opening peritoneum a foul smelling free fluid was encountered in peritoneal cavity. Further examination revealed a small perforation through wall of cæcum about half inch from base of appendix. In fact the entire cæcal wall for two to three inches from base of appendix was found to be inflamed and œdematous. The appendix was bound down by adhesions and slightly congested. As is our custom in all such cases, the general cavity of the peritoneum was packed off by iodoform gauze and a drain of the same character inserted to seat of perforation. No attempt was made to close perforation. Wound remained open. The appendix was undisturbed. Convalescence was rapid and uneventful. Drainage removed ten days after operation.

Second Operation, November 7, 1908.—Seventeen days after first operation adhesions separated. Appendix removed by the usual method employed by us of subperitoneal amputation. Wound closed without drainage. Patient discharged, cured, November 21, 1908.

This case undoubtedly belongs to the class which older authors, spoke of as typhlitis. Strangely enough we encountered two other cases shortly after the one reported, which closely resembled it. In all of these cases the cæcum was the primary focus of inflammation, the appendix remaining almost free from disease.

RETROPERITONEAL LYMPHOSARCOMA

Case IX.—S. S., aged 16 years, rivetmaker, white.

Chief Complaint.—Comes to hospital complaining of tumor of abdomen.

Family History.—Mother died of tuberculosis.

Past Medical History.—Negative.

Present Illness.—Patient states that until one month ago health was very good. About that time began to tire easily at work, and noticed that he was losing weight. Three weeks ago had an attack of severe pain in abdomen, which he attributed at the time to an indiscretion in diet. But this pain has since been more or less constant, though not quite so severe as when first appeared. Has lost fifteen pounds in weight. No cough and no night sweats. For the past two weeks has known of a tumor of the abdomen, about which pain centres.

Physical Examination.—Rather emaciated white boy. Skin clear. Superficial glands are not palpable in neck, axilla, or groin. Chest—on auscultation breathing is rather harsh, and there are a few fine crackling râles heard at apex of right lung; otherwise, lungs are clear throughout. Heart, normal. Abdomen—scaphoid; no free fluid; on deep palpation a mass can easily be felt in umbilical region; mass is irregular, cannot be moved, and does not fluctuate, somewhat tender to pressure. Pulse, regular, 85 per minute, temperature 100°.

Blood—leucocytes 9500. Urine—negative. No tubercle bacilli found. X-ray shows intra-abdominal tumor, not connected with stomach or intestines.

Clinical Diagnosis.—Retroperitoneal sarcoma.

Operation, May 25, 1909.—Incision in midline for three inches above umbilicus. No free fluid in peritoneal cavity. Two distinct nodular masses felt retroperitoneal, one removed and cavity packed with iodoform gauze. Abdominal wound closed. Tumor removed, resembled, very much, enlarged lymphatic gland. Subsequent to operation received Coleys' toxins and X-ray on alternate days.

Pathological Diagnosis.—Lymphosarcoma.

Patient's condition did not improve subsequent to operation. Left the hospital after two weeks, against advice. Returned to hospital July 15, 1909. Abdomen was markedly distended and he was more emaciated and very weak. July 16, 1909—abdomen tapped and a large quantity of milkish white fluid obtained. Fluid quickly reaccumulated and patient succumbed on tenth day of readmission. Tumor had increased in size about one-half, and evidently had obstructed chyle duct, this explaining color of free abdominal fluid. Postmortem was not obtained.

GALL-STONES—CHOLECYSTITIS

Case X.—Mrs. R. H., white, aged 37.

Chief Complaint.—Comes to hospital complaining of "indigestion," abdominal pain—slight jaundice.

Family History.—Negative.

Previous Medical History.—Negative.

Present Illness.—Has had "indigestion" for years. Says that food distressed her from one-half to one and one-half hours after eating, and has suffered from accumulation of gas in intestines. Has also had eructations of acid. Seven weeks ago, December, 1908, had first attack of acute abdominal pain, starting in right hypochondriac region and being referred to back and right shoulder. Pain was severe enough to "double her up," and was forced to stay in bed three or four days at that time. Vomited once during this attack. Has subsequently had several similar attacks, last one week ago. Skin has been slightly jaundiced lately. No chills.

Physical Examination.—Patient is quite a stout woman. Skin slightly jaundiced. Tongue coated. No enlargement of superficial lymph-glands. Chest—lungs are clear throughout. Heart normal. Abdomen—slightly distended; upper right rectus muscle is rigid and palpation, under edge of liver, shows tenderness; no

tumor mass palpable; liver dulness begins at fifth right interspace and extends to costal margin.

Urine—negative. Blood—leucocytes 12,000.

Operation, January 5, 1909.—Ether anæsthesia. Incision to right of right rectus over gall-bladder. No free fluid in peritoneal cavity. Gall-bladder somewhat injected. On opening gall-bladder large number of small stones removed, these stones closely resembling "Caviar." There was marked cholecystitis. Gall-bladder drained by means of rubber tube sewed into place by catgut sutures and fastened to parietal peritoneum. Closed by layer sutures.

Patient made an uninterrupted recovery and was discharged from hospital June 23, 1909, cured.

This case is cited as one representing a class of cases, which we are learning more about almost daily at our clinic. It has been our experience that a great many cases of so-called "indigestion," "sour stomach," and "biliousness," etc., when carefully examined, have revealed a history strongly suggestive of gall-stones as the chief etiological factor. Small "bird-shot" stones and a marked cholecystitis are usually revealed at operation. A simple cholecystostomy, draining the gall-bladder from ten days to two weeks, produces most satisfactory results in just such instances.

CARCINOMA OF PYLORUS

Case XI.—T. D., male, white, 29 years of age, miner.

Chief Complaint.—Comes to hospital complaining of nausea and vomiting, abdominal pain.

Family History.—Negative.

Past Medical History.—Typhoid fever when 19 years old. Three years ago, while working in a mine, fractured three ribs and right arm.

Present Illness.—Admitted to hospital May 2, 1909. Two years ago, August, 1907, had a severe attack of diarrhœa and was very ill for one week, being confined to bed. Did not pass any blood from bowel at that time, though he had as many as twelve to twenty stools a day. Patient states that he lost considerable weight then, perhaps twenty pounds, and has never since regained it. In February, 1908, began to suffer from a dull boring abdominal pain, starting in epigastrium and moving toward left hypochondrium.

This pain would usually become aggravated by taking food and would always be worse from one to two hours after eating. On the average of once in two weeks, would vomit a large quantity of partly digested food, vomitus being more "bilious" than "coffee grounds" in appearance. Has never vomited blood but thinks that, at one time, six months ago, stools were blood stained. In spite of rigid dieting and medication, symptoms have grown progressively worse. Has lost altogether thirty pounds in weight, and pain is more or less constant now.

It is interesting to note that patient's occupation required him to put his entire weight behind a drill, the handle of which he placed in the "pit of the stomach." At times it even became necessary for his brothers to lend their weight to his in pushing against this implement. They were forced to lean against him, so that pressure against his abdomen must have been considerable.

Physical Examination.—Patient is an emaciated, very anæmic, man; of average height. Mucous membranes, as well as skin, are very pale. Superficial lymph-glands are not palpable. Chest—lungs clear throughout. Heart normal. Abdomen—slightly distended; upper one-half of both recti muscles are rather rigid; marked tenderness to the right about two inches above umbilicus; no tumor felt; liver dulness extends from sixth interspace to costal margin. Extremities emaciated. Temperature 100°, pulse 90, respiration 20.

Clinical Diagnosis.—Carcinoma of pylorus.

Stomach Contents: Total acidity 30; free HCl none; combined HCl .05; lactic acid, a trace; Oppler Boas not found.

Blood: Hæmoglobin 21 per cent.; erythrocytes 1,460,000; leucocytes 8000.

X-ray shows mass at pylorus with dilatation of stomach.

Urine: Negative.

Operation, April 24, 1909.—Incision in midline of abdomen, four inches above umbilicus. No free fluid found in peritoneal cavity. On examining pylorus, hard indurated mass was found. Glands along lesser and greater curvature found to be enlarged, there were nodules in liver. Posterior gastro-enterostomy performed by "no loop" method, anastomosing stomach to jejunum. Wound closed, layer sutures.

Patient made good operative recovery and was discharged from hospital two weeks following operation. The clinical diagnosis was confirmed, this being the youngest patient operated upon for carcinoma of the stomach at our clinic.

GASTRIC ULCER

Case XII.—A. G., 56 years old, Russian, carpenter.

Chief Complaint.—Comes to hospital complaining of nausea and vomiting; pain in abdomen.

Family History.—Brother died of tuberculosis; otherwise family history is negative.

Past Medical History.—Had typhoid when 13 years old. Between 25 and 30 years of age had a severe attack of abdominal pain, was confined to bed for three weeks at that time and vomited small quantity of blood. Again when 42 had a similar attack, also vomiting small quantity of blood at this time.

Present Illness.—Began four years ago. Patient states that while drinking warm water he felt something "give way" in his stomach, causing him a violent attack of abdominal pain. Was confined to bed for ten days, and also had several vomiting spells at that time, but no hæmatemesis. During the past four years there has rarely been a time longer than two or three days during which he has been quite free from abdominal pain. This pain seemed to centre about two inches above umbilicus, and from there would radiate to right hypochondrium and back. Pain was aggravated by taking food and seemed to be worse from one-half to one hour after eating, when very often vomiting would bring relief. Has been vomiting more frequently during past two or three months and three weeks ago vomited all night, this vomitus containing half a glass of blood. Thinks that he is now twenty pounds below average in weight. Has never passed any blood by bowel.

Physical Examination.—Emaciated, white man. Cheeks sunken. Tongue coated. Superficial glands not palpable. Chest—lungs are clear throughout. Heart—heart sounds are distant, soft systolic murmur at apex. Abdomen—small epigastric hernia; abdomen tender and somewhat rigid in upper one-half; no tumor mass palpable; liver dulness from sixth interspace to costal margin;

right complete reducible inguinal hernia; extremities emaciated; superficial arteries slightly sclerosed.

Urine: Trace of albumin, no cast.

Blood: Hæmoglobin 80; erythrocytes 4,250,000; leucocytes 8000.

Stomach Contents: Total acidity 60, reaction acid; free HCl 0.15; combined HCl 0.11; lactic acid not present; Oppler Boas bacilli not found.

Clinical Diagnosis.—Ulcer of stomach. Old perforation (?).

Operation, April 15, 1909.—Ether anæsthesia. Incision in midline above umbilicus. On opening peritoneum, no free fluid found. Examination of stomach revealed old healed ulcer of lesser curvature with adhesions to liver. Posterior gastrojejunostomy performed. Abdomen closed, layer sutures. Patient vomited twice on third day and once on fifth day of convalescence. Otherwise, recovery uneventful.

CHRONIC INTESTINAL OBSTRUCTION—POSTOPERATIVE ADHESIONS

Case XIII.—A. S., white, female, 37 years of age. Referred to hospital complaining of obstinate constipation and abdominal pain.

Family History.—Negative.

Past Medical History.—Negative.

Present Illness.—Was operated upon for acute appendicitis March 4, 1906. Patient states that she had a great deal of abdominal pain following her first operation. Does not remember whether appendix was removed at this time or not, but states that her wound was drained for six weeks. Was reoperated February, 1907. At this operation "a part of her intestines were removed." Since her last operation has suffered continuously from abdominal pain, limited as a rule to the right lower quadrant, and becoming very sharp at intervals. Obstinate constipation has troubled her greatly, bowels not moving at times for five or six days, and then only by enema. She has vomited occasionally; has never vomited blood nor passed blood by bowel.

Physical Examination.—Well developed, rather obese woman, Skin clear. Superficial glands not palpable. Chest—lungs are clear throughout. Heart sounds distant, no murmurs. Abdomen—no distention. In right iliac fossa to right of McBurney's point

there is a mass, perhaps the size of a lemon. There is some tenderness on pressure over mass. No fluctuation. Scar over McBurney's point. Extremities normal. Temperature 99, pulse 90; respirations 18. Urine negative. Leucocytes 10,500.

Clinical Diagnosis.—Chronic intestinal obstruction, probably due to either intra-abdominal adhesions or abscess.

Operation, May 22, 1909.—Ether anæsthesia. Oblique incision to right of former scar. On opening peritoneum a mass of small bowel found matted together and adherent to parietal peritoneum. In attempting to free adhesions the gut was torn; promptly repaired by Lembert sutures of Pagenstecher linen. Entero-anastomosis performed by direct suture uniting small bowel proximal and distal to adherent mass. Wound closed with small gauze wick drainage to pelvis.

Patient made an uneventful recovery. Bowels moved on fourth day following operation and almost daily thereafter.

PAPILLOMA OF BLADDER

Case XIV.—J. P., white, 43 years of age, tailor. Patient comes to hospital complaining of frequent bloody micturition.

Family History.—Negative.

Past Medical History.—Negative.

Present Illness.—Patient states that for past year he has been passing bloody urine. When first noticed one year ago urine would only be blood-streaked perhaps once in two weeks; did not attach any particular importance to this condition until about one month ago. Since this time urine is practically invariably bloody and urination is much more frequent than formerly. Patient has to rise from four to eight times at night. There has never been any pus in his urine. Thinks that his feet and ankles have been swollen slightly at times during the past year. Has had no headache. Denies ever having had gonorrhœa or syphilis.

Physical Examination.—Rather anæmic. Skin pale. Superficial glands not palpable. Chest—lungs are clear throughout. Heart sounds are normal. Abdomen—negative. No arteriosclerosis. Blood pressure 130 m.m., systolic.

Cystoscopic Examination.—Shows small pedunculated tumor attached to anterior wall of bladder.

Fig. 11.

Anastomosis of median nerve.

Fig. 12.

Anastomosis of both ulna and median nerves complete.

Operation, January 5, 1909.—Ether anæsthesia. Suprapubic incision. Bladder incised through space of Retzius and an aluminum coated speculum introduced. Small pedunculated tumor found attached to anterior wall and removed by cautery. Bladder drained by rubber tube drainage for two weeks subsequent to operation. Urine perfectly clear subsequently though there was some difficulty in healing suprapubic fistula.

Pathological Report of Tumor.—Papilloma.

TRAUMATC INJURY OF FOREARM—NERVE SUTURE

Case XV.—J. C., male, white, 20 years of age. Comes to hospital complaining of wrist-drop (right) and wasting of muscles of right hand.

Family History.—Negative.

Past Medical History.—Negative.

Present Illness.—September 7, 1907, right forearm and wrist cut obliquely by a circular saw. Hemorrhage not severe at time of injury. Wound sutured at once and subsequently healed by primary union. Immediately noticed anæsthesia of palmar surface of right thumb, index and ring finger. Also noticed tendency of little and fourth fingers to contract. Two months later noticed wasting of muscles of this hand, palmar surface, which has steadily increased. Sensation on dorsal aspect of hand has remained undisturbed.

Physical Examination.—General examination negative. Local examination of right forearm, wrist, and hand shows a scar running obliquely for four inches from wrist fold, ulnar side, to base of metacarpal bone of thumb. There is marked wasting of lumbricales and muscles of thenar and hypothenar eminences. Little and fourth fingers remain flexed, other fingers and thumb extended. Sensation practically absent on palmar aspect of thumb, index, and ring fingers. Slightly impaired on palmar aspect of little and fourth fingers. Entire right forearm slightly smaller than left.

Operation, February 18, 1909.—Ether anæsthesia. Incision made from middle of palmar surface of hand to lower third of forearm. Careful dissection revealed severed ends of both ulnar and median nerves. Severed ends of median, each end presenting a bulbar swelling or neuroma, were first isolated and neuromas

removed. The nerve was then spliced by lengthening the proximal end. This lengthening was accomplished by dividing the proximal end through one-half of its circumference.

In a like manner the ulnar was sutured. The wound was then sutured, and the hand and forearm bandaged on a palmar splint. The wound healed by first intention. The case has not been heard from since leaving the hospital, so that it is impossible to state whether or not there has been actual improvement. The case is reported simply as a mechanically successful anastomosis of severed nerves seventeen months after original injury (Figs. 11 and 12).

EPIPLOITIS FOLLOWING THE RADICAL CURE OF HERNIA

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A short time ago I published a case of epiploitis following the radical cure of an inguinal hernia, in the *New York Medical Journal*. As this interesting lesion has not been extensively described, I desire in this paper to discuss the subject as fully as possible.

Epiploitis following the radical cure of hernia is more apt to occur in those cases where the omentum has undergone considerable manipulation during the operation, or when portions of it have been resected. The frequency of this complication is rather difficult to determine, some surgeons never having seen it, while others have seen it occur as frequently as once or twice in several hundred herniæ operated on by them. It should, however, be recalled that in hospital practice, there is difficulty in following the ultimate results, so that there are probably a number of instances of secondary epiploitis which have never been recorded, either for the reason that they occurred some time after the operation, or because they were mild in character. Nevertheless, it may be admitted that this is a relatively infrequent complication when one considers the large number of herniæ operated on and the few instances of epiploitis.

It is a matter of common knowledge that the omentum, like all other tissues, can become infected and suppurate. An epiploitis has been observed a number of times following removal of the adnexa, the appendix or after intestinal perforation. But the principal question to be discussed here is how infection of this organ takes place in instances of the radical cure of hernia. It is quite natural to think in the first place that the infection may have been brought directly by the hands of the surgeon, but in reality few believe in this. For that matter, an epiploitis following an operation for hernia may not make itself manifest for

months or years later and, from this very fact, the possibility of a direct infection is rather doubtful, because, were this the case, the affection would appear very much earlier. Then, again, in most recorded cases, the operative results were satisfactory, the wound healing by first intention.

I believe, however, that direct infection is possible and, in the first place, I would consider the ligatures. Many writers have incriminated silk, and in point of fact, in a large majority of cases, this material has been used. Out of 26 cases silk was employed 18 times and catgut only 8 times. Then, again, in suppurating epiploitis the silk ligatures have been found in the pus, the symptoms disappearing as soon as they had been eliminated. During operations the silk ligatures have been found included in a mass of omental adhesions and surrounded by a certain quantity of pus.

Since silk does not become absorbed it may be regarded as a foreign body remaining in the midst of the tissues, where it may set up an inflammatory process and favor the development of infection, which, otherwise, would have been properly taken care of by the organism. This is all the more possible because silk is difficult to sterilize. This material always contains large numbers of pores from which it is difficult to dislodge the bacteria that later on may give rise to epiploitis. On the other hand, silk is covered with an organic layer which increases still more the difficulties in its sterilization. Consequently, there is no doubt but that silk may be incriminated, but its mode of action still remains to be demonstrated.

Whether or not it transports the bacteria, whose virulence becomes awakened after a varying length of time and under various influences, directly into the tissues, or when aseptic it infects the omentum at a later time by contact with a neighboring suppurative lesion, are questions to be considered. Primary infection by the silk has been upheld by many eminent men and the difficulty of sterilizing braided silk is a plea in favor of this theory. Generally speaking, it is not doubtful that infection takes place at the time of the operation (Morestin).

The late development of the symptoms of epiploitis are rather adverse, I believe, to the theory of primary infection, but I would

point out that postoperative epiploitis does not always occur so late as many would lead us to believe. Out of 36 cases the commencement of the process was late in only 9, *i.e.,* in about 25 per cent.; twenty-seven times the symptoms appeared anywhere between 2 and 30 days following the operation. In the majority of them, it developed in from 20 to 30 days, and this period of time represents that spent in bed after the operation for hernia. I, therefore, believe that in these cases, which are the most frequent, the omental suppuration began quite early, but gave rise to no subjective sign while the patient remained in bed and the symptoms only manifested themselves at the time the patient resumed his ordinary life. Then, again, as I shall show in speaking of the treatment, rest in bed alone is quite sufficient to cure the process in many cases and from this fact, it is not at all unlikely that by remaining in bed, the early symptoms of the affection were attenuated. This much having been said, the argument founded on the tardy appearance of the omental phenomena loses the greater part of its value. Therefore, I am of the belief that primary infection from the silk takes place and this is due to the difficulty in its sterilization and its action as a foreign body.

Other surgeons uphold the view that the infection takes place secondarily and although not of frequent occurrence, secondary epiploitis may appear from four to six months or even three or five years after the hernia operation. Under these circumstances the hypothesis of a primary infection which has remained latent all this time should give way to another theory, far more simple and more nearly in conformity with the facts of the case. I refer to the influence of a secondary infection, either by the blood or through some other way, which becomes localized at the point where a foreign body exists. A ligature becomes secondarily infected when, placed on an organ with septic contents, such as a tube, a ureter, or an omentum infected by a long residence in a hernial sac, it finally cuts through the organ, placing it in direct contact with its septic contents. The tardy appearance of the symptoms would favor the theory of secondary infection of the silk ligatures. A certain number of observations certainly would uphold this theory, but, nevertheless, primary infection, in my way of thinking, is by far the most common.

In some reported cases the omentum had been ligated with catgut, but here secondary infection can hardly be admitted, because the ligature had become absorbed, but its infection at the time of operation must be admitted quite as much as in the case of silk. To demonstrate this fact I would refer to a case recorded by Schnitzler of a man who died a fortnight after a laparotomy from acute tuberculosis. The operative wound was perfectly aseptic, but a drop of pus was found on the omentum at a point where two catgut ligatures had been applied and in spite of this there were no symptoms of infection after the operation. Primary infection of the catgut must consequently exist, but when one is dealing with an epiploitis appearing several months or even years after the operation, some other mechanism must be invoked.

It may happen that a patient who is nervous, alcoholic or simply unmanageable, may displace his dressings after an operation for hernia, thus infecting his incision, so that, if an epiploitis should occur later on, the question arises whether or not this is due to the above fact. In one case the dressings became soaked with urine after an operation for hernia and ten days later there were symptoms of epiploitis, while in another instance epiploitis occurred the day following a soiling of the dressings. Cases of this kind are not numerous enough to justify the conclusion that the infection of the omentum was due to the patient himself. And then again, why should the infection choose the omentum rather than some other tissue? Is it because the ligated and resected omentum is at this time in poor condition? I believe that in these cases the omentum already is the possessor of the bacteria of infection, which, perhaps, would have remained in a latent condition for a more or less long period of time, or would never have given rise to manifestations of infection, but which were brought about from the movements of the patient and infection of the surface of the wound.

It would be quite an injustice to attribute exclusively to the operator all the complications that may arise in the omental stump. Very often the omentum itself contains the bacteria of infection, when, for example, it has been the seat of inflammatory attacks of recent date. This is ordinarily the case in instances of old

epiploitis. The organ has undergone changes, it is thickened, hard and resistant, its aspect as well as its structure is different from the normal, and it adheres to the sac or neck of the hernia. In point of fact the omentum contained in a hernia is often far from being in a healthy condition. Among the reported cases, only 31 give details as to the condition of the omentum found in the sac. In 14 of these the omentum was adherent 6 times, in 3 it was hyperæmic, adherent and strangulated, in 2 it was twisted, in 1 gangrenous, in 1 it was hyperæmic and hemorrhagic, while in 1 it was infiltrated. These various lesions are occasionally the result of strangulation and may be aseptic, but such is not always the case, because they may be due to bacteria coming from the intestine by way of the adhesions of a localized peritonitis or by way of the blood. The epiploitis, consequently, antedates the operation and the bacteria of the omentum sometimes, in spite of an extensive resection, will carry the noxious influence which they already exercised in the sac, into the abdomen. For this reason I believe it most probable that an adherent and chronically inflamed omentum must, to a certain extent, be held responsible for the accidents to which it may give rise.

As to whether or not resection of the omentum during a hernia operation is the cause of a future epiploitis, it may be said that this resection necessitates considerable manipulation and the use of ligatures, all of which multiply the chances of an infection. It is generally admitted that the omentum defends the abdominal cavity by destroying the bacteria introduced into the peritoneum, and Cornil and Carnot have demonstrated the part played by it in regeneration of the tissues and abdominal viscera. Milian has pointed out that the omentum is a mobile membrane which combats intra-abdominal suppurative processes, either by covering them and thus limiting their extent, or by giving issue to large numbers of leucocytes. Given these facts, it has been asked whether or not there was not a certain amount of danger in reducing the size of the omentum by resection and transforming it into a stump filled with ligatures.

Carnot states that, if the omentum is resected, a new tissue may arise in its stead, usually adherent to the neighboring parts. Here we have consequently an epiploitis after resection experi-

mentally produced. This is decisive and one should attribute to the resection a considerable part in the development of consecutive epiploitic accidents. But it would be rash to conclude that during a radical cure of hernia one should abstain from resecting the omentum contained in the sac, a point which I shall consider with more detail later on.

Little is known as to the bacteria producing secondary suppuration of the omentum. The colon bacillus has been found and this is not at all to be wondered at since it is the ordinary bacterium of the intestine and may reach the omentum either by way of the blood or by peritoneal adhesions. In one case the staphylococcus albus was found.

In considering the pathogenesis it will be noticed that I am inclined to believe that most of the opinions enunciated have their share of correctness and perhaps for this reason I may be considered too eclectic, but, as I have pointed out, sometimes the secondary epiploitis appears shortly after the operation for the hernia, while in other instances it may arise several months or even years after the operation; that in some cases catgut was used, while in others silk; in some cases the omentum was found healthy at operation, while in others it presented various lesions. Consequently, given such varying circumstances, it seems to me quite impossible to invoke a single theory as the only one which is correct and, on the other hand, if one considers the extreme diffusion of the bacteria, their tendency to insinuate themselves everywhere and to take advantage of all the ways offered them, such as the blood or lymphatics, gland ducts, and solutions of continuity, it must be admitted that when one wishes to determine the origin of a given infection, it would be quite illogical to assign such and such an entrance to the exclusion of all others.

Epiploitis following a radical cure of hernia makes itself evident clinically by a tumefaction which anatomically corresponds to the inflammatory products in the stump. These products may be purulent collections, adhesions to neighboring organisms or hard sclerosed tissues. Pus, adhesions, and the formation of sclerous tissue, are the three final outcomes of an inflammatory process.

In a certain number of instances one or the other of these

elements predominates so distinctly that one may distinguish three principal anatomical types,—namely, *the suppurative, the adhesive,* and *the sclerous.* In other cases these elements are combined in various ways, thus forming the *mixed types.* Out of 23 observations I find that the suppurative type was present 5 times, the adhesive type 5 times, the sclerous type 3 times; pus included in a mass of sclerous tissue 6 times, pus and adhesions 3 times and the three conditions united in 1 case.

After having opened the peritoneal cavity (when one is dealing with the suppurative type of epiploitis), as soon as the omental stump is reached, a fairly large purulent pocket is found, which, upon incision gives issue to a thick creamy pus.

This collection may work its way towards the surface and open through the abdominal wall and for this reason, when operating, one may occasionally reach the omentum at various depths. The purulent pocket is included in a mass of adhesions, which protect the peritoneal cavity, but they are not numerous and the pocket forms the principal pathological process.

The omental stump may be transformed into a central fibrous mass from which converge adhesions to various parts of the abdominal wall, colon, stomach, or intestinal coils. These adhesions being generally numerous and solid are a great danger to the patient. The loops of the small intestine may be agglutinated together by a mass of adhesions and in one case they formed a cuff, thick and hard in consistency, around the colon. Then, again it may be a loop of intestine which has become stretched across an omental stump which is adherent to the abdominal wall. And lastly, in another case, a loop of intestine was found drawn forward by firm adhesions uniting it to the abdominal wall.

In the sclerous type of epiploitis, the lesions found are particularly interesting. The organ is transformed into a grayish mass, bleeding easily, very hard and sclerous, adherent to the abdominal wall. The omentum, parietal peritoneum and the musculoaponeurotic layer may be involved by this sclerous infiltration. In this case, one is dealing with an abscess of the omentum, which, on account of its extreme induration, can be compared to a chronic phlegmon and in many cases it is with great difficulty that any focus of pus can be found.

On the contrary, in other cases, after having dissected through this resisting tissue, pus may finally be found and here we have one type of the mixed form. In one case a thick sclerous tissue composed of omentum was exposed, in the midst of which, after careful examination with the finger, no focus of pus could be discovered. Consequently, a second incision was made which exposed a similar condition of affairs, but, after having broken this up to a considerable extent, a purulent focus was found, giving exit to about an ounce of pus. This form is frequently met with and what should be particularly noted is, that in the mass of sclerous tissue, a small quantity of pus may be found. The purulent collection usually is in inverse relation to the size of the inflammatory tumor.

Very frequently one may encounter a second mixed type presenting both these elements, and in this class may be included two cases where the purulent pocket emptied into the intestine, both patients rapidly recovering. Consequently, it must be admitted that the omental adhesions were sufficiently strong and numerous to protect the peritoneal cavity. In a case reported by Schnitzler the purulent, sclerous and adhesive types were found combined. The patient presented a secondary epiploitis, which went on to suppuration, the pocket emptying into the bladder, this being made evident by analysis of the urine and cystoscopy. Under these circumstances the adhesions protected the peritoneal cavity. By rectal examination a mass was detected which in all probability represented the adhesions which walled off the tract followed by the pus from the omental stump to the bladder. In this case the cavity was opened and drained and part of its walls resected.

Microscopical examination in cases of epiploitis has not been resorted to frequently. I have referred to infection of the omentum by the colon bacillus and would repeat that in other cases the staphylococcus albus has been found in the pus. In one case a microscopical examination of the omentum was made, bits of which had been removed during operation in a case of sclerous epiploitis. The preparation was stained with hæmatoxylin-eosin. The blood-vessels were dilated and gorged with blood, while around them was a marked proliferation of embryonal cells which

almost completely filled the meshes of the connective tissue and formed large nodes in these meshes. In two places one found extravasated blood corpuscles in the midst of these embryonal cells, forming a true hemorrhagic infarct. In other words, the ordinary lesions of inflammation were present. In the second piece removed, similar lesions were found, but more accentuated. There was vascular congestion with a proliferation of embryonal cells and an extravasation of blood corpuscles forming a hemorrhagic infarct. In certain spots at the boundaries of the foci of cell infiltration were found bands of fibrous tissue, while at other spots the cells were poorly limited, stained with difficulty and appeared to be foci undergoing necrosis.

The explanation of the mechanism of the lesions may be given as follows: every epiploitis is a suppurative process at the commencement and should a second operation be resorted to on account of it, one will usually find small abscesses in the centre of the omental mass. I would recall Schnitzler's case of a man operated on for hernia with resection of the omentum; a fortnight later he died from acute miliary tuberculosis, and at autopsy a drop of pus was found in the omental stump. I would also refer to the case reported by Reynier of adhesive epiploitis, where, at the lower portion, the omentum formed a mass adherent to the gut. It was removed for a malignant tumor, and, when incised, a silk ligature was found surrounded by a small collection of pus. Consequently, it may be said that an epiploitis often commences by one or several small purulent foci developing around the material used for ligatures.

After this, certain cells give rise to the formation of adhesions, which partly protect the peritoneum and partly fight against the inflammatory process. Two antagonistic elements are constantly present, namely, the pus and the omental adhesions, and according to the one which is most prominent, one will be dealing either with the suppurating or the adhesive type of the affection. The formation of adhesions represents the reaction of the omentum against the initial suppuration, but it happens that the omentum may react otherwise; the blood-vessels dilate, bands of fibrous tissue form little by little, embryonal cells replace the fat vesicles of the membrane which soon becomes a very hard

sclerous mass, and the sclerous type is formed. This is a slow and only slightly active defensive resistance, but quite sufficient to control a torpid infection arising from bacteria of low virulence. It is the same type of reaction that is observed in certain forms of chronic phlegmon and actinomycosis, which are also examples of attenuated infection. In certain cases the adhesions or the hard sclerous tissue, although not completely destroying the suppuration, may at least arrest its progress and limit the purulent collection. Thus may be explained the mixed forms where one finds a pus focus and adhesions.

In the adhesive form the adhesions arrest the progress of the suppuration, but if they become extensively developed, they may give rise to complications in the gut. If they do not become too extensively developed, we then can understand those cases of epiploitis which get well without any operation, because they neither go on to suppuration, nor do they give rise to intestinal obstruction and we here have a development of adhesions sufficient to combat the suppuration, but not enough to produce intestinal obstruction.

In going over a number of cases which have been reported, it would seem to me that there is quite a uniformity in the symptomatology of secondary epiploitis, so that a good outline of these symptoms may be given. In spite of a few exceptions there exists a first stage, which varies, during which the patients present many points in common so that a description can easily be made. When this period is passed by, important variations occur in different cases, but at this time the progress of the affection has come near its end. After a radical cure of hernia where resection of the omentum has been resorted to, the postoperative convalescence appears satisfactory and the incision heals by first intention. Then the patient has a rise in temperature, but does not suffer; occasionally, however, a slight infection of the wound may be noted and slight pain may be present, but these incidents are of little import and do not indicate the accidents which are about to occur. Usually it is shortly after the interference, from one week to thirty days, that the intra-abdominal pain commences. It is in the first place localized around the umbilicus and on the side upon which the operation has been done. Slight at first, it is only felt

after eating, or when the patient moves or coughs, but it soon increases in frequency and intensity. It may also extend to the hypochondrium, iliac fossa, etc., and even to the lumbar region or the thighs. In other cases it appears intermittently and at regular intervals, disappearing only to recur again. It is sometimes so intense that morphine may be required, or it may be the only indication for operating.

Various digestive disturbances make their appearance; the tongue is coated, the appetite greatly diminished, while a painful spot is present in the region of the epigastrium. Nearly always nausea is met with and vomiting, which may be the first symptom noted, occurs frequently. In some cases, infrequent to be sure, fæcal vomiting may occur, indicating a peritoneal reaction. Constipation is usual and is accompanied by abdominal distention.

A few days after the commencement of these various symptoms, a tumefaction appears, usually in the upper portion of the abdomen, in the right or left hypochondrium, according to the side on which the omentum was resected. It is rarely found in the iliac fossa or hypogastrium, because it arises from the free border of the omentum. Now, as resection has taken place on this border, the latter no longer extends down to the pubis, but will usually be found in the neighborhood of the umbilicus. It is of common knowledge that, after resection of the omentum, the stump recedes quite high up into the abdominal cavity, and, consequently, the inflammatory mass will be found more often above the umbilicus and very rarely below it. In some few instances, however, it may occupy the iliac fossa or hypogastrium.

The tumefaction is indistinctly limited, but usually is the size of an adult hand. Later on it may increase in size and extend up under the costal border and down into the iliac fossa but not beyond the median line. This hard, tense mass, which is generally painful on palpation, is located directly behind the abdominal wall, glued, so to speak, to the parietal peritoneum. The skin covering it is normal in color and movable over it, as are the various layers composing the abdominal walls. If the patient is made to move so that the abdominal muscles contract, the mass will still be found separated from the abdominal walls. Later on, however, this condition may no longer exist, the omental stump

becoming very adherent by the development of adhesions or from the presence of a purulent collection which is making its way outwards. Then, again, it may be an infiltration slowly extending to and involving the various layers of the abdominal wall.

As to the mass itself, its mobility over the underlying structures is variable. Sometimes it follows the respiratory movements and appears free, at others it may be immovable. All this depends on the length of the omental stump and the number of adhesions present. Occasionally, a local tumefaction may be seen and in some cases is very marked. If the purulent collection is going to open externally the skin over it presents the ordinary characteristics met with in an abscess. The operative cicatrix, which is usually in a healthy condition, may occasionally present a small fistula giving exit to a seropurulent discharge, which finally gives exit to the ligature.

These various symptoms necessarily bring about disturbances in the general health. The pain, which is sometimes very severe, causes insomnia, which, with the vomiting and anorexia, brings about loss of flesh, to such an extent that in a few cases, a malignant neoplasm was thought to be present. The patients become weak and are obliged to remain in bed, while in some cases there is a peritoneal expression, accompanied by a small pulse, great thirst and restlessness. A rise in temperature is met with in nearly all cases, sometimes reaching the normal after a few days, in others remaining high during the entire evolution of the infection. In some cases there is a slight evening rise with an occasional increase, coinciding with an accentuation of the other disturbances. Generally speaking, the chart follows the intensity of the functional symptoms.

Pain, digestive disturbances, tumefaction, fever and a general malaise are the elements encountered in nearly all cases of secondary epiploitis, but, although this affection almost always presents the same symptomatology, the same can not be said of its evolution. For example, the tumefaction when once formed, undergoes several very different evolutions. It may disappear slowly by rest and medical treatment, the time varying from several weeks to several months. In other cases the omental mass, instead of progressively diminishing, increases in size, forming

adhesions with other points of the parietal peritoneum and viscera, particularly the small intestine, colon, and stomach, so that symptoms of intestinal obstruction arise and occlusion results. This serious type is fortunately infrequent. Finally, we have the third type which is common, in which a purulent pocket forms.

In cases of simple plastic epiploitis, the symptoms are limited and their intensity diminishes little by little. A cure of these cases requires time. The abdominal pain retains the character that it had in the beginning, occurring intermittently, or there is a continuous dull pain with spontaneous exacerbations. Little by little its intensity diminishes, but it remains as long as the omental mass can be felt, and it then disappears with the latter.

Anorexia, very marked at first, is often less persistent than the other symptoms and these patients may have a good appetite when the tumefaction is still large and the pain rather acute. The same cannot be said of constipation which is present during the entire duration of the affection and requires the administration of daily enemata. A slight intestinal obstruction may be met with and when it becomes more manifest indicates that adhesions have formed. The omental tumor is usually about the size of an orange or a closed fist, but it may be considerably larger. It is always hard, dull on percussion, but retains a certain mobility. It sometimes becomes adherent to the abdominal wall, but that is all. It increases slowly in size, the abdominal wall becoming less tense, palpation is no longer painful, the tumefaction is more deeply seated, probably on account of the diminution of its antero-posterior diameter. Finally, only a few foci of induration can be felt and the process is at an end.

In this form the general health is not much disturbed; the patient may be somewhat thin and his strength diminished, while there are a few rises in temperature. In order to obtain a complete cure several weeks or months are usually necessary, but in the end resolution is certain and occurs without any incident. Simple plastic epiploitis, the most frequently met with, always has a favorable outcome.

In plastic epiploitis with adhesions, the pain is much more severe and constipation is stubborn, so that enemata and purgatives finally cease to have an effect. Finally, no gas is passed

so that operation must be resorted to. Vomiting and nausea may also be present and occasionally the former becomes fecal. In certain instances the omental mass may acquire very large proportions and instead of being limited to the hypochondrium and iliac fossa it may extend to the median line and even to the other side, while in some few instances it has been known to fill the entire abdomen. This naturally does not happen in all cases, but the omental mass is always very large in epiploitis accompanied by adhesions.

At the time the pain is most intense the abdomen becomes distended and on account of the pain the patient cannot eat and finally becomes greatly emaciated. The above-mentioned phenomena may finally end with intestinal occlusion so that interference is urgently demanded. An emptying of the gut, however, may suddenly end the symptoms of obstruction, but these may recur later and an operation will again be indicated. In epiploitis with adhesions giving rise to symptoms of obstruction it is better to operate at once.

In suppurating epiploitis the pain is sharp and over a large area, this being explained by the formation of the purulent collection, which, in order to develop, pushes aside its neighboring structures and disassociates the layers of the already inflamed connective tissue. When the purulent collection has reached its full development the pain diminishes and will totally disappear when the pus has become evacuated, either by operation or spontaneously.

Anorexia is common in suppurating epiploitis and is due to the infection of the organism; likewise vomiting is frequent and constipation usual.

The omental mass is quite large and hard and by palpation is more or less painful. Little by little it loses its firm consistency, becoming soft and pasty. After a variable length of time the point of fluctuation can be determined and then the diagnosis can be conclusively made. This is accomplished by the acute aspect of the affection and a rise in temperature, because fluctuation does not always occur or the purulent collection develops deeply, compressing and partially obstructing the gut; consequently the superficial fluctuation cannot be detected and there is complete absence of this symptom. Oftentimes in these cases the abscess empties

into the intestine, this being made evident by the presence of pus in the stools.

The temperature is usually quite high, following the ordinary course found in intra-abdominal suppuration. The pulse corresponds with the temperature and is small and thready. The patient presents great prostration and this with sweating, high temperature, and the character of the pulse, will usually permit one to make a diagnosis, even if fluctuation cannot be detected. As soon as the purulent pocket is emptied a cure usually takes place.

In some instances, before one has had time to interfere, the omental abscess will open spontaneously through the abdominal wall. The result is an irregular opening, giving issue to pus which will last a long time and the resulting abdominal fistula will hardly ever be recovered from excepting by surgical treatment.

When a diagnosis of a suppurating epiploitis has been made it should be treated by free incision and drainage. The pus soon ceases to come away, especially when the ligatures are eliminated at once. The operative wound closes by second intention and recovery soon follows. It should also be pointed out that this operation is far from being easy in all cases, because the search for the purulent focus is sometimes extremely difficult on account of the presence of adhesions, and one may be obliged to make other incisions in order to reach it. Recovery is usual even under these circumstances.

Occasionally the abscess may open into the small intestine or colon. The patient experiences a severe abdominal pain, followed by an imperious desire to go to stool and he is very much astonished to find the fæces mixed with pus and blood. An immediate improvement occurs, the various symptoms disappear and recovery is rapid.

The abscess may also open into the bladder, as has been reported in one instance.

Simple plastic epiploitis and suppurating epiploitis are met with with equal frequency. Epiploitis with adhesions is rarer. Out of 45 cases I find simple plastic epiploitis twenty times, suppurating epiploitis nineteen times and epiploitis with adhesions six times, thus making 45 per cent. for the first, forty-two per cent. for the second and 13 per cent. for the third type.

The diagnosis presents various difficulties. The fact that a radical operation for hernia has been done is of great importance, particularly if the epiploitis began one, two or three weeks afterwards, at the time when the patient was about to leave his bed, or is actually out of bed. It is certain that the greater the time between the development of the symptoms and that of the operation, the less likely is one to find a relationship between the two. It should be recalled, however, that secondary epiploitis often arises a long time after the operation, months or even years having elapsed.

The first symptom of the affection is that of a peritonitis in some cases, while in others the onset is insidious and without any particular character. There is pain of varying types and constipation which is common to all abdominal infections and, therefore, cannot aid the diagnosis. Careful palpation will often localize exactly the tumefaction and if the latter has not already contracted adhesions the mass will be found lying directly under the abdominal wall. Its location can also be confirmed by percussion, because the dulness will show that the tumor lies between the wall and the gut. Percussion will also show that the tumefaction is distinct from the liver or spleen and consequently it will become evident that one is dealing with the omentum. If its hardness is noted and if it is known that the patient has undergone a radical cure of hernia, there can be hardly any doubt as to the nature of the affection.

These secondary epiploitides may be mistaken for abdominal tumors, but I will merely mention a few points in the differential diagnosis. Even if one suspects that the condition of affairs is a postoperative complication, a diagnostic error is still possible, because the tumefaction may be taken for a large hæmatoma due to the falling off of a ligature applied to an artery in the omental stump. But this accident occurs very shortly after the operation, not infrequently before it is finished. The internal hemorrhage which results should be at once detected by the ordinary symptoms of this complication. Even a severe hemorrhage, however, may cease spontaneously, thus giving rise to a hæmatoma. The latter will not be present unless the symptoms of internal hemorrhage have already preceded it.

Neoplasms or abscess of the abdominal wall are quite accessible to the exploring hand and are mobilized by the contraction of the muscles of the abdominal wall. It must be noted, nevertheless, that a suppurating epiploitis frequently tends to invade the superficial structures, giving rise to suppuration in the sheaths of the recti, so that at this time palpation alone is of little diagnostic value.

An intestinal growth is always more deeply seated, giving rise to a large tumor, abdominal distention and melæna, while digestive disturbances are much more marked and the cachexia more accentuated.

One can hardly mistake a secondary epiploitis for a tuberculous peritonitis, because in the latter affection the lesions are more scattered and omental masses and distended intestinal coils are felt here and there, so that by percussion, dulness and tympanism alternate over the abdominal surface. Likewise the abdomen is very sensitive to pressure and ascites is present. It is likewise frequent to encounter pulmonary lesions of tuberculosis.

When the tumefaction is situated low down in the iliac fossa, for example, other diagnostic errors are possible. A malignant tumor of the pelvis may be suspected, but in this case the evolution is much slower than an epiploitis, the pain is severe and extends along the sciatic nerve, there is unilateral œdema, varicose veins and so forth. Then, again, if the patient is carefully watched for a few days, no doubt is longer permissible.

And lastly, if the secondary epiploitis is seated on the right side and the tumefaction fills the iliac fossa, one may suspect an appendicitis and a differential diagnosis is really very difficult to make.

There is no pathognomonic sign of epiploitis following the radical cure of hernia and each symptom belongs to many other affections so that individually they are of no great semeiological value, but taken as a group with a consideration of the progress of the affection, they will be of great value. Therefore, it is the knowledge that a radical cure for hernia has been performed on a patient that is of the greatest usefulness. It must be admitted, however, that there are some cases particularly difficult to diagnosticate and one should always look for the scar of the hernia incision if unfamiliar with the history of the case.

I have already pointed out the long duration of simple plastic epiploitis, but, on the other hand suppurating epiploitis, with spontaneous opening of the omental collection of pus through the abdominal wall may result in a fistula, which, on account of its exceedingly long duration, becomes a true infirmity for the patient. Then, again, besides the inconvenience from the constant discharge of pus, necessitating a frequent change of dressings, the patient may also be seized with more serious accidents. The abdomen becomes distended, vomiting and constipation arise and the temperature goes up. These disturbances which last for two or three days recur quite frequently and these frequent repetitions, along with the fistula, render the life of the patient extremely miserable. The most serious complications of epiploitis arise when adhesions are present and here we will meet with phenomena of intestinal obstruction which may be extremely marked and recur so frequently that in the end cachexia results. Finally, after several attacks of obstruction, intestinal occlusion manifests itself. The life of the patient is seriously compromised if operation is not immediately undertaken. To all these troubles may be added general peritonitis. At all events, such conditions are not frequent, because of the well-known tendency of an epiploitis to form a tumor or adhesions which will limit the purulent collection and protect the peritoneum.

The opening of the purulent collection into some abdominal organ, such as the intestine or bladder, has in all cases, as far as I am aware, resulted in a cure. Such an ending, however, is not exempt from danger and should be included among the complications of this affection.

In more than 50 per cent. of the cases, secondary epiploitis is recovered from by rest in bed and medical treatment. The cure is sometimes long in being attained, but is practically certain. In the other cases surgical interference is necessitated and, if undertaken in time, will improve the outlook as far as the prognosis is concerned. Therefore, when one is dealing with a suppurating epiploitis, a large incision should be made to give exit to the pus and this being followed by free drainage, will, in all probability, prevent the formation of a fistula, and likewise ward off the accidents which might arise from the opening of the abscess into

the deeper structures. Out of 46 cases that I have collected there were two deaths, in other words a mortality of a little over 4 per cent.

The prognosis is only serious when the epiploitis is accompanied by adhesions, but, fortunately, this is rare. Under these circumstances, there is danger of repeated attacks of obstruction and these will finally lead to cachexia, even if they do not end in occlusion. Even under these circumstances surgical interference may save the patient, especially if the adhesions are few in number. On the other hand, if they are very dense and multiple, the success of the operation will be more or less doubtful, but, nevertheless, it should be undertaken as it gives the patient his only chance.

Resection of the omentum during the radical cure of hernia is, as I have pointed out, one of the causes of secondary epiploitides. For this reason the question has arisen as to whether or not it should be resected during the operation. If the omentum is found hyperæmic, indurated and adherent in the hernial sac, it should, by all means, be resected and this resection *should be carried out as high up as possible in healthy omental tissue.* Resection should also be done when the omentum in the hernia is overloaded with fat and cannot be reduced. If it is found healthy and the mass in the sac moderate in amount, it should be, nevertheless, resected, because it is one of the principal etiological factors in the formation of hernia. At the same time the resection is advantageous, inasmuch is it diminishes the abdominal contents.

Certain surgeons have upheld that resection gives rise to abdominal pain and secondary hemorrhage, as well as secondary epiploitis. Then, again, many are the writings which have demonstrated that the omentum protects the abdominal organs and defends the abdominal cavity in cases of infection and, therefore, it should not be meddled with. Nevertheless, I am firmly of the opinion that all these reasonings do not invalidate the necessity of resection of the omentum in all cases where it seems necessary, but that resection should be done with all proper precautions. For instance, the omentum should never be ligated *en masse,* because this produces traction in the axis of the transverse colon and results in the slowing of the circulation in the omental stump. The omentum should be divided into several pedicles, each ligated with

a single ligature. The ligatures should be applied as far as possible in the least vascular portions of the omentum, and when reducing the organ into the abdomen, it is better to commence with the shorter pedicle. As to the choice of the ligature I particularly commend kangaroo tendon. Silk should be absolutely discarded, and catgut should not be used unless one sterilizes it oneself, for I am unfamiliar with any make now offered to the medical profession that I would trust.

Secondary epiploitis is occasionally due to various changes arising in the omentum which is contained within the sac. These changes are more particularly encountered when strangulation and gangrene have taken place and especially in aged subjects who have been possessors of their hernias for a long time. They have worn a truss for a more or less lengthy period and from its pressure it has given rise to various lesions in this organ. On the contrary, in young subjects the omentum found in the hernial sac is usually healthy and is present in moderate amount.

In considering the treatment it may be said that a secondary epiploitis will usually develop in the form of a simple plastic inflammation. Consequently, the patient should be kept in bed and ice bags applied to the abdomen. Constipation should be controlled and, if the pain is severe, narcotics may be given. It is important to make a daily examination of the tumor, pulse and temperature, as well as of the intestinal functions. Very often everything will quiet down by this treatment, although it may take a certain length of time. For this reason operation must not be too hastily considered.

I will now discuss those circumstances which indicate surgical interference. If one is dealing with a suppurative epiploitis, the purulent collection must be opened. Two cases present themselves in practice. In the first case the tumefaction is distinctly fluctuating and no difficulty is encountered in cutting down on the pus. The only rule to observe is to make a sufficiently long and deep incision and drain the cavity thoroughly. The suppuration will soon decrease, the wound will contract and a complete cure is not long in being attained. But, if the collection to be emptied is deep-seated, fluctuation is not made evident and here one must be entirely guided by the temperature and the pain. In

these cases operation is often difficult and requires great delicacy, but, when the pus has once been found, the patient will recover, though convalescence will here be much longer.

In epiploitis with adhesions all symptoms of intestinal obstruction arising therefrom require early surgical interference. If laparotomy is done early one will usually find a few omental adhesions and by resecting them, a cure results. In other cases one is called upon to interfere later on in the process and then the intestine will be found lost in the mass of adhesions and is readily wounded if great care is not taken. When the adhesions are very numerous and tough, one cannot hope to remove them and, under these circumstances, all that can be done is to break up their points of attachment on the parietal peritoneum.

In all cases of epiploitis with adhesions the interference should be undertaken early, because as time goes on, they increase in number and thus render the operation more difficult. As to occlusion of the intestine and peritonitis, complications which in truth are rare, I have little to say. Abdominal fistula should be treated by laparotomy and resection of the fistulous tract. Knowing that these fistulæ are the result of a purulent focus kept up by the presence of foreign bodies, particularly by silk ligatures, this focus must be reached, carefully cleaned out and the ligatures if possible removed. They never become eliminated themselves. Other difficulties may result from a sinuous tract followed by the fistula which not infrequently presents bends and branchings. The same is true of the consistency of the surrounding tissues and the depth of the purulent pocket. In several cases recorded in literature, a favorable result has been said to have been obtained by the introduction of a tent of laminaria within the fistulous tract, but I cannot commend this, as it appears to me most unsurgical and recalls to one's mind the innocent, yet glorious days of the infancy of gynæcology. In other words, the treatment of epiploitis and its complications must be strictly surgical and by that I mean the surgery of the present day.

COLOR PHOTOGRAPHS IN RELATION TO SURGERY *

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For many years, ever since it has been possible to fix the ray of light on a sensitive surface, it has been the dream of the scientist to be able to reproduce the colors of nature by the aid of the camera. The ray of light could be divided by the prism, the diffraction rings of Newton, and the soap-bubble film. Why, then, could it not be gathered in such a way by a sensitive plate as to show color.

True it is that the production of light-sensitive plates with the silver salts came years after Newton's time. But the experiments of this scientist, working in the dark, foreshadowed not only the eventual production of a light-sensitive plate, but also the recording of color by that plate. Niepse, Daguerre, and other scientists spent their lives in experimentation with the haloid salts. Although they gave us the marvel of the photograph, yet they failed to fix the color of the flower on the plate. Many years elapsed between Newton and Lumière; but without the deductions of the one the results of the other would probably not have been possible. In the lapse of time between these two men many scientific ideas had been recorded, and gradually the possibilities of color photography had been evolved.

The making of color photographs at the present time is probably still in its infancy; although beautiful results have been obtained by several processes, the methods of production are cumbersome, and not always true to nature, and they have many disadvantages; either the process is very complicated, or it is still quite imperfect, and the final result can be modified in many ways. Hence there is a lack of definiteness to any process heretofore devised—that is, the result can be modified, and must often be modified, to suit cer-

* The photographs herewith shown were made in the Photographic laboratory of the American Oncologic Hospital, Philadelphia, with a Bausch & Lomb Zeiss-Tessar lens, 8 x 10, and of about ten inch focus. The photo-micrographs were made on their Zeiss photo-micrographic camera, using a 16 m m. objective, and a No. 2 compensating eyepiece.

tain common conditions; or else it is modified by circumstances over which we have, as yet, but partial control.

The history of color photography is very brief, but extends over many years. Each step evolved by the early workers has been taken advantage of later by some other investigator, who was thus enabled to carry the matter a little further along. Thus, Zenker, working from Newton's interference of light waves on the soap-bubble film, was able to formulate certain deductions; Lippman, in turn, carried the deductions a step further, and succeeded in obtaining photographs in true colors, when viewed by white light under certain conditions. Thus the Lippman process by interference has been the first of a series in the production of a color plate direct by the camera.

The Lipman interference process utilizes for the production of color a very thinly coated emulsion plate, very poor in silver salt; the plate is backed up by a film of metallic mercury in mechanical contact, which acts as a reflector. The rays penetrate the film and are reflected by the mercury back of the plate and traverse the film a second time. In doing this they interfere with, or cross, each other, and the ray of white light is resolved into its component colors by its action on the molecules of silver salt contained in the film. When the plate is developed, fixed, and dried, and viewed in a given position in white light, a very vividly colored picture is the result, which is produced by the white rays being decomposed by the molecules of silver and thus delivered to the visual apparatus in their primary colors. But it must always be viewed in a fixed direction to exhibit the colors, as at any other angle the ray of white light is not decomposed by the particles of silver in the film, and the colors are not produced.

Among those who have contributed much to this process are the Lumière people, who have succeeded in producing some very fine results thereby. But it has proved, up to the present time, a process of scientific value at best, and requires for its production the utmost care of a trained mind, and the resources of a physical laboratory. It is of scientific interest in that it is a most important step in color photography and may be utilized later.

R. W. Wood, of Wisconsin University, has produced photographs that show color by a diffraction process. The principle is as

follows: A ray of white light when decomposed by the prism forms several colored rays, the most important of which are red, green, and blue. These, recombined in proper proportion, again produce white. These several rays are represented by varying wave-lengths —a short wave forming the blue end of the spectrum while the longer length will produce the red end, etc. By having sheets of plate glass ruled in fine parallel lines it is possible to produce these colors. By varying the rulings per inch the red, green, and blue can be produced at a certain position by the diffraction of light by these several rulings, each ruling causing the spectrum to occupy a position further removed from the centre of the plate. The exposures are made through these ruled screens, on glasses coated with bichromated gelatin. Three exposures of the same object are made, each through the ruled screen for red, green, and blue. Two exposures are made on one plate and one reversed exposure on the other. They are developed and fixed by washing in water, and dried. When superposed, although each glass bears but a transparent gelatin film, the whole will present the colors of the original object, if viewed by white light at a given angle to the centre of the plate, the ray of light being decomposed into its primary colors by each of the superimposed films.

Prof. Joly, of Trinity College, Dublin, has given us the first definite colored photograph, and one from which the Autochrom has probably been devised, as they are similar in many respects. In the Joly photograph the color is seen by looking through and not by looking at the picture as in the Lippman plate. The photograph is taken through a screen which is very closely ruled in vertical parallel lines of color, red, green, and blue (of such proportion as, when viewed at a distance, will blend into white), and indefinitely repeated across the plate. It is evident that this ruling must be very fine so that it will not be too apparent to the ordinary vision. Plates with lines $\frac{1}{200}$ inch wide have been ruled commercially. This ruling must also be very exact, for if there is any appreciable variation the viewing of the picture through the complimentary screen will be more or less impaired. The exposure is made through this three-color ruled screen. It is manifest that the resulting negative will have its surface infinitely divided by lines corresponding to the taking screen. This virtually gives us a nega-

tive that contains three different exposures or views, any one of which can be viewed separately by appropriate means. From this negative a positive is made by contact. When the positive is finished it is backed by a viewing screen similar in ruling to the taking screen, but in complementary colors, which colors are slightly different from those of the taking screen, as will be noted later.

It is manifest that the success of this process depends on the production of the taking and the viewing screens in sufficiently fine rulings, and in such manner that they can be produced commercially with accuracy; for, as one taking screen is sufficient for any number of exposures it is necessary that a viewing screen shall be secured permanently to each positive, in order that the register may not be afterwards disturbed. The production of color in the finished product depends entirely on this register, as any overlapping of the lines of the positive on the viewing screen will fail to produce the color. The placing of the positive in proper relation to the screen, and securing it in that position, requires some skill; hence it is apparent that the great advantage of the Autochrom plate is to be found in its fixed register, which prevents all displacement of the positive on the screen.

To Ives belongs the credit of giving us a true color process, and to him we are indebted for a vast amount of scientific facts in regard to color photography. Whether the Ives pictures are viewed by the kromoscope, or are produced as in the superimposed pigment prints, the results are nearly perfect. But the process has the disadvantage of being very complicated and entirely beyond the ordinary worker in photography, although commercially it is of the most pronounced value. The process requires very exact though entirely feasible methods to produce the best results.

The process consists in taking three separate negatives of the same subject from the same viewpoint through screens of red, green, and blue color; these negatives are previously sensitized so that they are best adapted to their special portion of the spectrum. From these negatives half-tone plates are prepared, and by superimposing these plates and printing them in transparent pigment of the complementary colors, the true color of the print is produced. Or, if the views are to be used in the kromoscope or lantern, a transparency is made from each negative, and these are viewed or

projected through the appropriately colored screen for each picture, and the three are blended into one view, thus combining the three colors in such proportion as to produce the colors possessed by the original object. Whoever has seen these views in the kromoscope (which is a binocular instrument, requiring six views of the subject) has been struck with the accurate color-rendering of the resulting stereogram, which almost approaches perfection. Were it not for the extreme complication in the making of these views the instrument would undoubtedly find great favor with many workers.

The Autochrom process follows to an extent the Joly process, in that the picture is taken through a three-color screen to produce the negative; from this is made the positive, which is, in turn, viewed through the same three-color screen. But there is this difference: that while the Joly screen is a ruled one, the Autochrom screen is made up of closely deposited three-color starch granules, and the positive is formed of the remains of the silver emulsion not utilized in the formation of the negative. The Joly positive is temporarily fixed over the three-color screen, while in the Autochrom it is permanently fixed, thus insuring a perfect and permanent register. On this account the Autochrom picture is complete in itself, both the three-color screen and the positive being superimposed on the same plate.

Though seemingly intricate, and requiring many steps to complete the Autochrom picture, the process is fairly simple, and is easily within the grasp of any person familiar with the underlying principles of photography.

In the preparation of the plate the glass is covered with a layer of starch granules, very minute, and colored red, green, and blue. These colors must bear a certain relation to the spectrum, so that when intimately mixed in certain proportions and viewed by transmitted light they are seen as white. These granules are fixed permanently on the plate in a varnish which is not disturbed by the after processes. This constitutes the screen of primary colors. The plate is then covered with a thin layer of slow panchromatic silver emulsion, on which the final photograph is made. The panchromatic emulsion is sensitive to all the colors of the spectrum, thus ensuring a true gradation of color along the whole length of the spectrum.

The making of the photograph is fairly simple. The plate is placed in the plate-holder with the glass side towards the lens (the reverse of the ordinary process). Either in front of, or back of, the lens a special ray filter is placed; this filter is of a color that retards the rays from the violet end of the spectrum. In the ordinary plate the blues and violet hues show whiter than the reds or bright orange tints. These latter colors are in reality brighter colors to the eye than are the blues. By the use of the ray-filter the red end of the spectrum has an opportunity to impress the light-sensitive film to a greater extent, with the result that the colors assume more nearly the proper color-value as they appear to the eye; that is, the reds and yellows appear bright, while the blues are more subdued. The daylight ray-filter is a brownish-yellow color, darker than the ordinary filter. As the filter is probably aniline dye it will have to be watched—it should be tested spectroscopically to detect slight changes; any change of color will, of course, mean a disturbance in the quality of the light that reaches the plate, with a corresponding defect in the color of the picture. The light passes from the image, through the lens, ray screen, three-color screen, and impresses itself on the sensitive film. The exposure is about sixty or seventy times as long as is necessary for the ordinary plate. For this reason it is necessary to have a good light.

The development is done twice; the first is with a pyro-ammonia developer, carried on for two and a half minutes in absolute darkness; it is washed, and placed in a bath of acid permanganate of potash solution until all the silver thrown down by the first development is destroyed; the second development is done in daylight; this makes all the remaining silver salt sensitive to the second developer, which is done with dianol; all the silver is now thrown down as a black deposit. The colors can now be seen by looking through the plate. The second development has produced an image which is thin, and it must be intensified with nitrate of silver-pyro solution, which increases the brilliancy of the colors; what little silver there remains is now fixed out with hyposulphite of soda; after washing and drying it is varnished. It now presents all the color more or less perfectly, the degree of perfection depending on the quality of the light, freedom from scratches, fog, and other

blemishes that so easily interfere with the perfect result from these delicate plates.

This process, it should be understood, does not produce color; the color is all on the plate, in the shape of the colored starch granules before the picture is taken. Much of this color will have to be cut out or covered up in order that the remainder may be available. This is done by the silver in the second development, which performs two offices—it not only shuts out all the color not needed, but forms the black and white portion of the picture which gives shape to the whole. All the available color is seen through the clear spaces following the second development, which are formed by dissolving out the product of the first development, making, as it were, windows, each covered with an appropriate color granule. These windows are selected by the camera—the red ray from the object impressing itself on the sensitive film back of the red granule, and the blue ray back of the blue, etc. The part of the emulsion acted on by this light is precipitated by the first developer, and eaten out by the permanganate, thus leaving a hole in the film, which is covered by its appropriately colored window. It is seen from this that the three-color screen acts both as a taking and a viewing screen, and cannot be changed for a screen of complementary colors as is done in the Joly process.

The mixed colors are compounds of the primary colors, as explained in discussing the Ives process; thus, orange or yellow is formed by the mixture of red and green; purple by red and blue; white is a mixture of all three primary colors, in a definite proportion.

The fact that the Autochrom taking and viewing screen is the same, constitutes one of the errors of this process. In the Ives and Joly processes the viewing screen is made up of colors complementary to the taking screen; this is made possible by the fact that these screens are not a part of the picture, but are added to it. In the Autochrom process it is not possible to change the screens, as the film is superimposed on it. Hence it can be seen that if the screen is correct for taking it is incorrect for viewing, and *vice versa*. Assuming that red, green, and blue are the proper primary colors for taking, what will be the proper colors for viewing? On this subject there has been a great deal of work done by Ives and

Abney; their final results differ slightly, and in order that this may be followed out the following table is given:

Ives.

RECORD.	VIEWING.
Red	Prussian blue
Green	Magenta pink
Indigo blue	Lemon yellow

Abney.

RECORD.	VIEWING.
Red, vermilion washed with thin transparent blue	Apple greenish yellow
Emerald green	Magenta pink
Ultramarine blue	Greenish yellow

On this basis when we view the Autochrom picture instead of looking through a pink portion of the screen it is necessary to look through a red portion. This should produce the effect of making the picture look too red. As a matter of fact this really is the result, and the error of the Autochrom is that it usually looks too red. People viewing the picture have remarked on this point without knowing the reason for it. This defect can be compensated for to an extent by using a little lighter ray screen which will let through a little more blue, or by using a blue tint reflector; but it will not wholly remedy matters. This modification of the ray filter really must be made in photomicrographic work, as will presently be shown.

Barring this defect in hue and a few other minor points, the fact remains that the Autochrom process is the simplest for accomplishing color photography that has yet been worked out; the results, while not perfect, are such as will give nearly ideal photographs, which can be used in various ways. Their principal value is for teaching and for illustration; on these two points too much stress cannot be laid; for both purposes a color photograph is so far superior to a black and white one that it excludes all argument to the contrary. The time must come, and that soon, when these pictures (or those by some other process equally simple) will have a very important place in teaching institutions and in scientific publications. In no other way than in color can medical and surgical conditions be properly illustrated. The more expensive methods

which rely on the ability of the artist for color effects, and which are seldom perfect, will give way to the more accurate and less expensive methods of direct photography.

Much of the illustration by chromolithography when well done is above reproach; but there is chance for considerable error in the personal knowledge and judgment of the artist; in the cheaper grades of this work this has been very apparent, and many illustrations in color are practically worthless on this account. It will therefore be expedient to eliminate the inaccuracies of this method by a method such as the Autochrom process, which gives a fairly correct rendition of color, and which can be reproduced on paper by the three-color printing process.

As the success of the Autochrom plate depends largely on the condition of light employed, in a general way it may be said that the most satisfactory effects are obtained in a diffused daylight; direct sunlight usually produces shadows of too great contrast that are lacking in detail and the colors are not so well brought out. Nevertheless, on account of the character of some subjects and the necessity for short exposures, sunlight must be employed. The most perfect light is the midday light, for this is a purer light than can be obtained later in the day. Frequently an afternoon light will impart quite an undesirable yellow tint to the photograph. One other reason why the noonday light should be chosen is the closer approach to a standard of intensity which this light possesses; given a clear sky and a fixed hour there need be very few mistakes made in timing these exposures; this uniformity of time is more perfect at midday than it is latter in the day. In the afternoon it is extremely difficult to estimate the amount of actinic light available. Many of the light-meters on the market are of service, and when uncertain it is better to make use of one of them than to guess about the matter.

Failures with Autochrom plates are many. The plates do not keep well, and a fogged plate will work thin, or give no image at all, depending on the amount of the fog present. In hot weather the films are very liable to frill and sometimes strip. Various imperfections of film may appear as black spots in the finished picture. Breaks in the continuity of the film take on a very obtrusive green color, which no amount of retouching will obliterate.

Over-exposures give a flat picture with very little color. Reflections from surrounding objects may modify the color. Other conditions may arise that may render the production of these pictures anything but satisfactory.

In the accompanying photographs in color and in black and white it has been my endeavor to illustrate the advantages of a color process.

Figs. 1 (*Frontispiece*) and 5 illustrate a case of lupus vulgaris. Fig. 5 (left side) shows condition of patient on entering hospital. The disease is active; scales and crusts cover the whole area, which includes the opposite side of face. Several attempts were made to procure an Autochrom of this condition; but as the weather was hot, when the plates were giving a great deal of trouble, the attempt had to be abandoned after five or six failures. Finally at the end of six weeks, with the onset of cool weather and with a new lot of plates, an Autochrom was finally obtained (Fig. 1) of the opposite side of face. This shows a later condition of the disease, well advanced to recovery. It will be observed that nearly all the scales and crusts have disappeared, and that there is only to be seen the fairly uniform redness of the scar, except here and there along the thickened margin and over the ear, where the disease is still fairly active. Even though considerable areas of this inflamed base are shown in Fig. 5 this is not apparent as red color, but is simply depicted as dark or black areas, which manifestly does not indicate the true condition of the patient.

The error inherent in the ordinary plate of showing certain shades of red and orange (very bright colors visually) as black in the photograph is a very trying one. Even in the isochromatic plate this is not wholly overcome, and the violet end of the spectrum is shown out of its true proportion of hue as it appears to the eye; hence the great advantage of showing this condition of the patient in the true color over a black and white is here demonstrated.

In the average hospital of to-day a tremendous amount of clinical material passes under observation every year without an effort to have photographic or similar records preserved. This is greatly to be regretted, as all hospitals get, at times, very rare material which deserves a better fate than is being given it at the present time. There should be some systematized effort made to take

photographic as well as written records of all cases which present themselves, especially those with interesting pothological conditions. The expense of doing this need not be great; almost every hospital has someone on whom it can call who is competent to look after this work. The results achieved in a year or two will surprise all persons concerned; for in this time there has been accumulated a wealth of pictorial records which will be a perpetual fund of information for all who have access to them; and these records will grow more valuable year by year, and may eventually be of more interest than are the temperature charts or other clinical data. Certainly this must be apparent to any person engaged in teaching or in writing; no one can predict just when a photograph of a given condition will be needed; it may not be for years, or until a similar case for comparison appears in the clinic. We may greatly desire to possess at this time a photograph of the first case long since departed. It is not always the important case, either, that we wish to have; many of the minor cases make very interesting studies; for often a photograph or a series of photographs will illustrate a subject better than will an endless length of written description; especially is this the case when a judicious selection of the cases has been made, and some of them in color. It is not necessary to have all of them in color, for that would ultimately prove too expensive; but for the more important ones color is advisable, as we then have an additional element for comparison and study which we do not possess when all the subjects are in black and white.

When a patient presents himself to the observer a reasonable consideration of the case photographically will be as follows: (1) Photograph of the condition before treatment; (2) photograph of the gross specimen after treatment; (3) photomicrograph of the specimen; (4) photomicrograph of involvement beyond growth; (5) photograph of patient at various times following operation as interesting conditions present themselves. An arrangement of this kind will usually give us all the information we need, and will make up as comprehensive a history as the notes themselves, and will be of great value for future reference.

1. *The Condition before Treatment.*—This will show the exact location and appearance of the disease. If, for instance, it be a carcinoma of the breast, it will show the exact portion of the breast

PLATE II

Fig. 3.

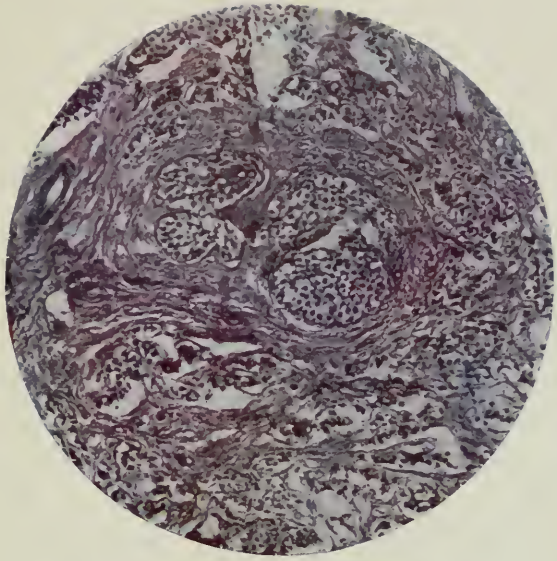


Photo-micrograph carcinoma of breast (× 100).

Fig. 4.

Gangrene of foot

involved; will indicate whether or not there is contraction of the nipple, and to what extent; whether there is an accompanying eczema of the nipple; and whether the nipple is hypertrophied; the situation of the diseased area; the comparative distance from nipple to axilla or nipple to growth; the tumefaction of the breast; and, if broken down, the exact size and appearance of the ulcerated area. If the arm is included in the view the occurrence of œdema will be seen; the axilla will show whether there is sufficient involvement to produce swelling; one general measurement noted will enable us to arrive at any other dimension fairly accurately. The growth may, however, be of such a shape, or bear such relation to surrounding parts, that one photograph will not be sufficient to give all the necessary points; in that case two or more may be needed to record the condition correctly. In some conditions pictures from several directions are required; and particular attention should be paid to obtaining the most important features and relations of the diseased area in the picture. All these are very important points, and a photograph will record them much more accurately than can a written description, which may vary greatly if taken by two different observers (Fig. 6).

2. *The Gross Specimen.*—After removal of a part other additional facts can frequently be recorded by the camera. In the series here shown the breast is seen (Fig. 2, *Frontispiece*) divided in a line drawn through the nipple and the diseased area. Here the diseased area is accurately shown, and a better idea of the retraction of the nipple can be had; the intimate relation between the outgrowth of the disease and the nipple must not be overlooked, and the contraction which occurs in the growth is especially in evidence, pulling the nipple towards the diseased area, and causing at times a furrow between the nipple and the surrounding skin. The nipple being thus bound to the growth, any traction on the nipple will of necessity cause movement of growth in same direction. Whether or not the skin is adherent and moves with the mass depends entirely on the amount of involvement of subcutaneous fat and connective tissue; if the growth is deeply situated it can readily be seen that this classical symptom may not be present.

For the more careful study and comparison of this point it is well to make critical comparison between the Autochrom view

(Fig. 2) and the black and white view (Fig. 7), both of which are shown for comparison. In the colored view the underlying muscle, the diseased area, and the overlying fat and skin are all more clearly shown than they are in the black and white view.

3. *The Photomicrograph.*—All pathological tissue should be studied under the microscope. Under certain conditions a diagnosis can be made by general appearances; but in most cases it is necessary to make the final diagnosis with the microscope. That this is essential is being demonstrated daily, for the macroscopic appearance and the microscopic findings of a growth are often at variance, and at the present time conclusions have very little weight unless confirmed by a microscopical examination.

But we are not always in the laboratory with the microscope; hence it is well to record, in some way, the findings of the microscope for later study; this cannot be done better than by photographing what the microscope reveals. To be of service a photomicrograph must be well made, and this requires the employment of expensive apparatus, which is not in the hands of every worker. Even though it is not possible to acquire the best apparatus, this branch of photography need not be wholly neglected, as very fair results can be obtained by the use of medium-priced apparatus. The average quality of the photograph may be lower, but by care and perseverance a very good result can frequently be obtained. On general principles, however, the better the tools the better the work that is turned out, so that everyone who contemplates doing this class of work should have the best he can afford. We can then record in our photomicrographs precisely what the microscope shows.

Heretofore the one drawback to the photomicrograph has been the lack of color; the black and white plate does not always interpret the findings of the microscope correctly; for in the examination of a black and white print we have not the advantages of the differential staining to guide us, even though the form and arrangement of the cells are shown, by which alone much knowledge, indeed can be gained. But with the Autochrom plate we add the advantages of color; for fortunately the stain colors can be recorded with this plate in a remarkable manner. In the future this class of work will be largely produced on the color plate. Certain con-

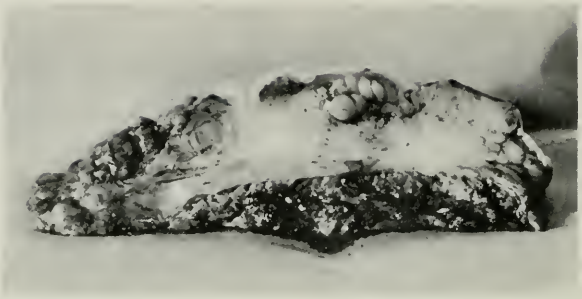
Fig. 5.

Lupus vulgaris.

Fig. 6.

Carcinoma of breast.

Fig. 7.



Section of breast showing carcinoma.

Fig. 8.

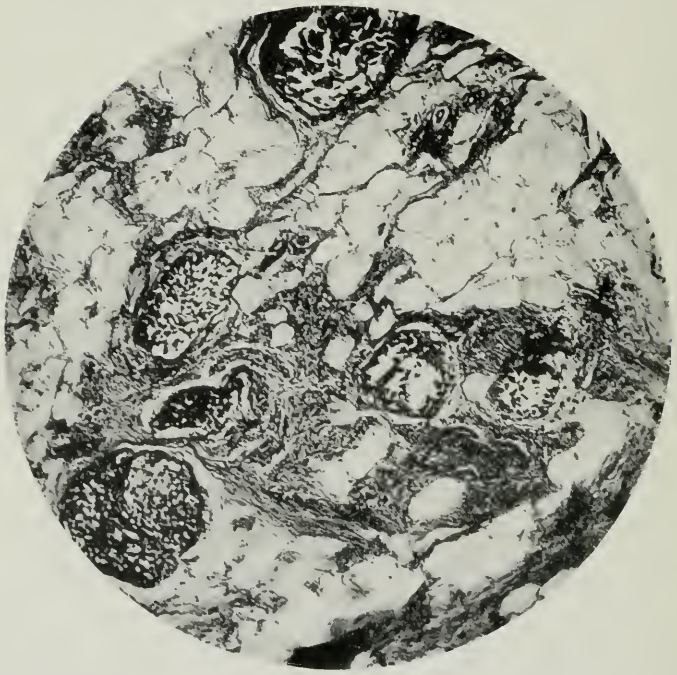


Photo-micrograph—axillary lymph-node from same patient as Fig. 3—carcinoma (× 100).

Fig. 9.

Postoperative involvement of chest by carcinoma.

ditions are met which must be correctly worked out that we may be the better able to achieve success in this line of work on Autochrom plates; these will be discussed later.

When we compare the Autochrom photomicrograph with the black and white we at once see that we are greatly aided by the staining of the specimen. There is one object sought by staining, and that is the differentiation of the tissues and cells. Therefore, when we can differentiate on the plate the several colors as well as can be done from the specimen we have made a distinct gain, and we derive all the advantages of reproducing these colors as well as the microscopic form of tissue on the color plate, and can demonstrate the structure on the plate quite as correctly as can be done with the microscope (Fig. 3). Therefore a photomicrograph of one or more portions of the growth should always be made, and this preferably should be in color.

4. *Involvement Beyond the Growth.*—This is as important as the growth itself, and requires as close study; it may show continuity with the growth, or extension beyond the visible area. It is necessary to determine definitely whether this tissue is identical with that of the growth, or is of some other nature—whether it is malignant or inflammatory. On this depends the extent of the operation or the necessity of subsequent operations; frequently a glandular involvement will show the same type of disease as existed in the primary lesion (Fig. 8); occasionally it may show simple inflammatory involvement; in either case a photomicrograph should be made of it for future reference, and it will be a guide to the necessity for future interference.

5. *Postoperative Photographs.*—These are among the most instructive pictures we can have; frequently, as in malignant disease, when an operation fails to be curative, the series of photographs taken from time to time after operation, as conditions change, form most valuable pictures for study (Fig. 9). No written description can compare with this method of making records. If some of these pictures can be made in color it materially adds to their value. For class-work these pictures should be systematically taken; for very often our clinical material changes in appearance from week to week and it is necessary to keep systematically and closely in touch with it. Such photographs should not be left until the case terminates one way or another.

The plan here outlined is not necessarily an inflexible one; modifications may be desirable in many instances to conform best with the individual conditions; but we do insist on the adoption of some definite plan of picture making for those who have the best interest of their students at heart, or for those who wish to report, in a systematic manner, their cases in print; otherwise we will continue to drift along in an uncertain way, and fall short of the high standard which we might otherwise attain.

A word as to the making of photomicrographs on the Autochrom plate is necessary. In a general way it encounters the same limitations as does the ordinary plate. The plate being very slow it is necessary to make an exposure about sixty times as long as is done in the ordinary process, which means from ten minutes to four or five hours, depending on the stain, the amplification, the light, etc.; these long exposures are made by the illumination of the Welsbach light, which is lacking in actinic rays; with acetylene or electric illuminants the time is much shorter.

Lack of absolute sharpness is a great drawback in the Autochrom; this is very apparent in the photomicrograph, where we expect to find very sharp detail; but this cannot be sharper than the starch-granule screen, which is coarse enough to be apparent to the unaided eye.

The correct exposure for the Autochrom is important; it should not vary beyond certain limits for the best results; when in doubt the best way is to ascertain a correct timing by actual trial on an ordinary plate, and increase this by sixty times, which will give very closely the time for the Autochrom under the same conditions.

The staining of tissues is important; any stain will be reproduced fairly correctly, but a good, clean stain with sharp contrast will give the most brilliant pictures.

The greatest difficulty is encountered in the use of the ray-filter; the regular Autochrom screen cannot be used as it will block out nearly all the violet end of the spectrum; if a Welsbach light is used (this being yellow) the screen must be greatly reduced, the amount depending on the color of the balsam in the mount; all balsam mounts yellow with age. A very yellow mount can be taken without any screen, while a clear, fresh one will need a screen. The selection of the proper color for the screen will greatly tax the

judgment of anyone, as on this it will depend whether our photomicrograph is to be too blue or too red; a dense screen will make it too red, while too thin a screen will make it too blue. The most desirable screen is a liquid one made of the aniline dye Aurantia, more or less diluted with water to meet requirements. A very serviceable and a more cleanly screen is made by fixing out unexposed lantern plates in hypo, washing, and staining with a solution of Aurantia of different strengths; by making three or four such plates a selection can be made to be used with any color of balsam. For average work a depth of color equivalent to about one-fourth the daylight screen will be approximately correct for an average mount.

We thus briefly describe the Autochrom process, and note its advantages and shortcomings. With all its faults it represents an advance far beyond anything we have had heretofore for scientific photography. There is no doubt but that in the near future it will be perfected and simplified, or some other process better adapted to our daily requirements will be forthcoming. Until this time arrives we may use with great benefit the Autochrom process.

THE USE OF ETHYL CHLORIDE AS A GENERAL ANÆSTHETIC

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The inhalation of ethyl chloride will induce general anæsthesia without the respiratory irritation of coughing, gagging and vomiting so often seen during the first and second stages of ether and chloroform anæsthesia.

With the patient lying in the supine position, breathing quietly and naturally as though preparing for sleep, the ethyl chloride is slowly dropped upon several layers of wide meshed gauze held about six inches from the face, and while the gauze is gradually approached to the face the dose of ethyl chloride is increased until the patient loses consciousness, when the gauze is folded and shaped about the mouth and nose in such a way as to exclude the air and the ethyl chloride given rapidly in the form of a spray. The patient will have reached the stage of unconsciousness without the least suggestion of suffocation, coughing or other irritating symptoms. Some time before the loss of consciousness there is anæsthesia to very severe pain and this stage may be employed for minor operations, the opening of abscesses, setting of fractures, dressing of painful wounds or many of the surgical procedures of five to ten minutes duration which do not require muscular relaxation.

When the ethyl chloride has been given rapidly or in large concentrated doses there is frequently an arrest of respiration at the time consciousness is lost, while tonic spasm of the masseter muscles and even general muscular rigidity or convulsions may occur. But if the patient is being carefully watched and the anæsthetic is removed at the first sign of this "forgetting to breathe" or the appearance of muscular spasm the respirations will be resumed and the muscular spasm relieved. If, however, the ethyl chloride is administered slowly, allowing for two to three minutes to the stage of unconsciousness, with the gradually increasing dosage and free admixture of air permitted by the gauze, a more careful observation

of the progress of the anæsthesia and a timely recognition of the danger line is allowed, with much less frequent occurrence of the respiratory arrest and muscular spasm.

As the anæsthesia progresses the eyeballs begin to roll and the pupils partially dilate, indicating that the patient has entered the second stage in which there is deep anæsthesia without muscular relaxation and frequently with muscular rigidity and spasm. It would seem that this is as far as the average patient can be safely carried, for after this stage the danger increases rapidly, the eyeballs become fixed, the pupils dilate widely and become immobile, the corneal reflex disappears and the face is flushed and covered with perspiration. This is undoubtedly the danger line beyond which the respirations insidiously become shallower with consequent deepening cyanosis, the eyeballs may diverge while the muscles are rigid, in clonic spasm or on the other hand flaccid.

The fatalities seem to result primarily from respiratory, and secondarily from cardiac failure, the heart continuing to beat for some minutes after the stopping of the respirations. Large and Brown [1] experimenting upon dogs found there was always a fall of blood pressure, which was preceded in a few cases by a slight rise, and they believe the respiratory failure follows a paralysis of the respiratory centre as a result of the lowered blood pressure.

Thus with ethyl chloride we can quickly and pleasantly carry a patient through the preliminary stages of anæsthesia, but it is rarely possible to obtain the muscular relaxation of the third stage of ether or chloroform, and then only by approaching to or going beyond the danger point. Therefore, when general muscular relaxation is desired other anæsthetics may be combined with it, thus avoiding the unpleasant features of the preliminary stages while obtaining the relaxation of the third stage of ether or chloroform.

The anæsthesia is started by dropping the ethyl chloride upon gauze and the ether or chloroform is introduced drop by drop upon the same piece of gauze at the period when consciousness is lost and while the dose of the second anæsthetic is rapidly increased that of the ethyl chloride is gradually decreased. If a sudden change is made from the ethyl chloride to another anæsthetic the patient will recover from the effects of the first intoxication before that of the second appears.

Though it is generally accepted that ethyl chloride will thus quickly and pleasantly induce anæsthesia there is still a great difference of opinion regarding its safety when compared to ether and chloroform. Hewitt places it between ether and chloroform, estimating the mortality from ethyl chloride at 1 to 10,000 and in the latest edition of his book quotes McCardie's figures of 1 to 3000. Luke [2] makes one estimate of 1 to 8000 and a short time later another of 1 to 150,000.[3] Both Luke and McCardie have had a personal experience in over 2000 cases without a mishap, and Herrenknecht [4] makes a similar report of 3000 cases, while Webster [5] recently reports 1800 cases with a clean record.

Such a varied difference in opinion would indicate the presence of other elements, independent of the anæsthetic, as causes of death and a careful analysis of the twenty-five reported cases certainly demonstrates them. Thus seven of them occurred during dental operations when the anæsthetics were administered by the dentist or an assistant. In eight cases the patients were in the upright position when the ethyl chloride was given. In seven of the cases where the method of administration is mentioned a closed or semi-closed inhaler was used and a mass dose of three to six cubic centimetres given.

The following is a short abstract of the fatal cases which have been reported in literature.

Case II.—A 12 months old child with diphtheritic laryngeal obstruction.

Case III.—This case, reported by Dr. Allen in 1903, was a colored man 28 years old with an incarcerated hernia. He had been vomiting freely but the vomitus was not fecal in character and his condition seemed good otherwise. During the change from ethyl chloride to ether anæsthesia he suddenly recommenced vomiting, bringing up large quantities of a clear fluid. This vomiting lasted from three to four minutes and the respirations were never resumed. An examination by the Surgeon, post mortem, failed to show any pharyngeal or laryngeal obstruction and he considered it a death due to the anæsthetic. There was no autopsy.

Case V.—A large healthy man, 24 years of age, with a huge submaxillary abscess. Several minutes after the removal of the anæsthetic and after the abscess had been opened, respirations

stopped so suddenly that it suggested acute laryngeal obstruction. At least four minutes later a tracheotomy was done without the re-establishment of respiration. At the autopsy there was found a marked œdema of the glottis with a relaxed fold of mucous membrane of the wall of the abscess so placed that it might have been sucked into the small air-passage remaining between the vocal cords.

Case VI.—A male 67 years of age, whose autopsy showed a very large mass of malignant cervical glands encroaching upon the lumen of the pharynx and larynx and involving the vocal cords.

Case XXII.—Ethyl chloride was chosen as the anæsthetic for a negro, 17 years of age, with an acute ischiorectal abscess, because of a harassing cough, profuse expectoration and the physical signs of a chronic consolidation of the left lung. He was placed in the lithotomy position, and though never thoroughly anæsthetized, received considerably more than the usual ten grammes, for the administration lasted over a period of more than ten minutes. Ten or fifteen minutes after the withdrawal of the anæsthetic there was a violent paroxysm of coughing, after which the respirations ceased and, though partially re-established by vigorous stimulation, artificial respiration and tracheotomy, the pulse became imperceptible after the paroxysm of coughing and the heart did not respond. This death was different from the usual ethyl chloride catastrophe in that the cardiac failure was primary.

Case XXIII.—A negro, 30 years old, while attempting a highway robbery one week previous to his admission to the hospital, received a load of buckshot in the lower part of the left axilla. He had remained in hiding all this time without medical attention and when he entered the hospital there was a large gaping wound in the lower portion of the left axilla and the physical signs of a general peritonitis and profound sepsis. While being placed upon the operating table his pulse became imperceptible and after receiving less than a gramme of ethyl chloride, given in the usual way, and before any operative procedure could be started, his respirations ceased. The autopsy showed a large wound of the left pleura and an empyema of the same cavity; a wound and empyema of the pericardial cavity; a wound of the diaphragm; perforations of the stomach and the intestines; and a purulent peritonitis.

Case XXIV.—An unmarried negress, 30 years of age, who was being treated in the medical wards of the hospital for *adiposa dolorosa* developed a Ludwigs' angina associated with a marked laryngeal obstruction. Incisions beneath the jaw opened the sublingual space and allowed a few drops of pus to escape. When the patient was placed in the dorsal position the laryngeal obstruction was considerably increased and after receiving about 5 grammes of ethyl chloride, given in the usual way, her respirations stopped, the pulse remaining unaffected, but they were quickly resumed after the removal of the anæsthetic and short artificial respiration. Twenty-four hours later, the œdema and laryngeal obstruction having increased, another operation was attempted, and as before, the respiratory obstruction was greatly increased when she was placed in the dorsal position and a very short time after the anæsthetic was started, cessation of respiration became complete, and respiration was never re-established, though a tracheotomy was rapidly performed. The autopsy showed an acute inflammation and œdema of the pharyngeal, sublingual and cervical tissues with œdema of the glottis.

Case XXV.—A young married negress with the diagnosis of tubo-ovarian abscess was given an unknown quantity of ethyl chloride preliminary to a proposed ether anæsthesia. After taking the anæsthetic for one or two minutes it was noticed that the respirations had ceased, and though the pulse could be felt for a short time after this it soon disappeared and cardiac stimulants together with artificial respirations were of no effect. There was no post-mortem examination and a physical examination made just before the anæsthetic was given was negative except for the presence of a loud systolic heart murmur without any signs of broken compensation.

The physical condition of Cases III, V, VI, XXII, XXIII, XXIV make it doubtful whether death would not have occurred with any anæsthetic and Cases XXIII and XXIV certainly would have died without any anæsthetic. Such statistics, compiled as these have been, from many sources are necessarily modified by the varying conditions of their administration, and the report made from the Pennsylvania Hospital,[6] in which all the ethyl chloride and ether anæsthesia given during a period of five and a half years are analyzed, would seem to eliminate many of these modifying

conditions and offer a fair comparison of the mortality of ethyl chloride and ether. It was found that five deaths occurred during the administration of ethyl chloride to 5575 cases and that 3 occurred during the administration of 5592 cases of ether. But even these statistics are distorted for it is a record of deaths occurring during anæsthesia and not of deaths due to the anæsthetic. The difficulty in defining the cause of death on the operating table seems to be avoided whenever possible, but it hardly seems fair to credit Cases III, XXII, XXIII, XXIV to the anæsthetic.

With such statistics the reason for the uncertainty of the safety of ethyl chloride will be appreciated, but it seems fair to consider any agent, which can produce deep anæsthesia in from fifteen to twenty seconds, and whose danger signs are so easily passed, less safe, in inexperienced hands, than either ether or chloroform. The number of cases reported by Luke, McCardie, Herrenknecht, Webster, and the writer's own experience of over 1000 cases, without fatalities, would indicate that skilled administration does influence the mortality.

Aside from its safety, Packard has recently called attention to the stiffness and even rigidity of the jaws of cases receiving ethyl chloride and which persist even though ether is subsequently given. This stiffness of the jaws in the early stages continues in many cases throughout the period of anæsthesia and limits its use in a field of surgery to which it would seem peculiarly adapted,—that is, the removal of tonsils and adenoids, and for which it was at first so extensively used in England.

CONCLUSIONS

1. The inhalation of ethyl chloride will induce general anæsthesia quickly and pleasantly.

2. Such an anæsthesia is peculiarly adapted for minor surgical operations not requiring muscular relaxation or extending over a longer period than ten minutes.

3. When used in combination with ether and chloroform the unpleasantness of the two preliminary stages is eliminated, while the muscular relaxation of the third stage of ether or chloroform may be obtained.

4. Though is seems impossible from available statistics to form

an accurate estimate of its safety, any agent that will produce deep anæsthesia in from fifteen to twenty seconds and whose danger signs are so difficult to recognize cannot be considered as safe as ether in inexperienced hands.

REFERENCES

[1] Brown: Laryngoscope, St. Louis, 1905, xv, p. 8838.
[2] Luke: Lancet, 1906, i, p. 1235.
[3] Luke: Lancet, 1906, ii, p. 1473.
[4] Herrenknecht: Munch. med. Woch., Dec. 3, 1906, liv, 2409.
[5] Webster: Surg. Gynæc. Obs., 1909, April, p. 402.
[6] Lee: Annals of Surgery, Nov., 1908.

Röntgenology

THE RÖNTGEN DIAGNOSIS OF PULMONARY TUBERCULOSIS

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The value of the Röntgen diagnosis in early tuberculous lesions of the lungs has been widely discussed and many claims have been made for it, some of which seem absurd, when it is considered that they can be substantiated by no other methods of physical diagnosis.

The discusion of its value in prætubercular and incipient stages of tuberculosis would be valueless, and this paper will be confined to its employment as supplementary and confirmatory of the physical signs adduced by other methods.

In this direction it is of value since it furnishes a permanent record with greater accuracy and detail than can be obtained by auscultation and percussion.

The Röntgen rays register upon the photographic plate the relative densities of tissues through which they pass. The amount of penetration varies with the quality of rays employed and should be varied to suit the tissues to be studied. Thus they can be made to show contrasts in bony structures, or between bones and the tissues surrounding them, or variation in the density of areas of soft tissues.

A negative to be of diagnostic value must be made with rays which will produce contrasts in the tissues to be studied. Thus in studying the lungs for tuberculosis a quality of ray and a length of exposure must be employed that will produce a contrast in soft tissue densities without entirely penetrating them. The thorax with its air filled lungs and dense mediastinal structures readily lends itself to the production of contrasts.

The technic which will show minute pathological lesions is dependent upon a rapidity of exposure which has only been recently developed, since the respiratory motion of the diaphragm and the

pulsation of the heart produce motion in the lungs. Suspended respiration and exposures made during that period increased the definition in the structure of the lungs, but exposures of one-fourth of a second or less are necessary to eliminate the motion resulting from the heart-beat.

Röntgenograms, to be of value as a basis for diagnosis, should be made under these conditions as they have been shown to produce increased detail which has materially assisted in the detection of the lesser pathological changes.

The value of a pathological examination under the microscope is dependent upon the ability of the microscopist to make a section which will show the tissue to be studied under the microscope, and to interpret it correctly. The diagnosis he renders is valuable in proportion to his technic, skill and experience in his special line of study.

The Röntgenologist's diagnosis is equally valuable in proportion to his technic, skill and experience in his work. A beautiful X-ray negative is valueless, unless it shows proper contrasts in the tissues to be examined, and the experience of the person who reads it measures the value of the diagnosis.

The tubercular process must have produced a macroscopic pathological lesion in the lung or the peribronchial glands before it can be shown in the Röntgen negative. Even then this method is incapable of determining the etiology of the change. The recognition of a pathological change has and will often lead to a more careful study of the case and result in the detection of the cause.

This method is therefore valuable in the early stages of tubercular disease because it adds definite evidence of pathological change to a clinical picture which lacked confirmation by physical signs. These added data often confirm an early diagnosis which could not have been made without it, though of itself this method could not form the basis for a diagnosis.

The nature of these confirmations will be better understood by reviewing the pathological expressions of pulmonary tuberculosis as seen in the Röntgenogram.

Lesions deeply situated within the thorax have been repeatedly shown when they had escaped percussion and auscultation, while the symptoms pointed to a tubercular lesion.

FIG. 1.

Tuberculous bronchial adinitis. Heart in diastole.

Fig. 2.

Tuberculous bronchial adinitis. Heart in systole showing cardiopleuric space.

Fig. 3.

Tuberculous bronchial adinitis with infiltration of right apex and elevation of diaphragm on that side.

Fig. 4.

Tuberculous peribronchial infiltration.

FIG. 5.

Tuberculous consolidation of right lung with cavities in upper and middle lobes. Pleura thickened over lower lobe. Infiltration and cavity in left upper lobe.

Fig. 6.

Consolidated left lung with cavity in apex. Superficial pneumothorax over left lung. Compensatory emphysema of right lung.

Thus infiltrated and even calcified peribronchial glands have been shown before the tubercle bacillus had been found in the sputum. In many cases where physical signs were absent and the bacillus present, with symptoms pointing to the disease, the picture has shown enlarged bronchial glands or deep seated peribronchial infiltration. Increased density in the apices of the lungs has often confirmed a physical diagnosis of impaired resonance and altered breathing, while a dulness on percussion which seemed superficial was shown to be due to a deep seated infiltration or commencing consolidation.

The study of evident areas of consolidation, softening and cavitation determines more accurately their situation and extent, while not infrequently deeply situated areas of consolidation and cavities have been shown that had escaped detection by other methods.

As has been said, the Röntgen rays take cognizance of the increase or decrease in relative density. Thus a marked contrast to the normal is shown between consolidation and compensatory emphysema, the latter being more readily penetrated than the normal lung.

A dilated bronchus can be shown for a similar reason, as in a recent case in which an area at the base of the right lung and in the median line posteriorly was supposed to be a localized empyema, but proved to be dilated bronchi that had been filled with a mucopurulent fluid.

The increase in density produced by a thickened pleura cannot be differentiated from an equal area of infiltration except by a stereoröntgenogram. The results of a pleurisy, as localized empyema or pyo- and hydropneumothorax can be distinctly shown. In one case a superficial area of pneumothorax overlay a consolidated and collapsed tubercular lung confusing the physical signs until its presence was detetcted by the negative. In another case a true hydropneumothorax of tubercular origin was demonstrated, while in many instances the effect of old pleuritic adhesions was plainly visible in alterations in the curve of the diaphragm.

The relative height of the diaphragm upon the two sides is in a measure an index of the relative capacity of the two lungs, but it is not a constant sign as many other conditions within the thorax vary the relative capacity of the lungs without altering the level of the diaphragm.

Besides the alterations in the lungs and pleura produced by tuberculosis the Röntgen rays will recognize pericardial effusions and thickenings of tubercular origin.

The relations of the heart and larger blood-vessels are often greatly altered by the changes in the lungs. The extent of these alterations has been clearly shown by this method of examination. Not only is the heart drawn over to the affected side by the contracting lung, assisted by the compensatory emphysema, but its axis is also often so altered as to place it in an anteroposterior position. The great blood-vessels and other mediastinal viscera are also displaced often so far that they lie entirely on the affected side beyond the median line.

Another pathological change frequently noted in the seventy odd cases recently studied is a calcification of the costosternal cartilages. While this may occur from other causes its frequency in tubercular cases is worthy of note.

The re-examination of tubercular cases and the accurate comparison which can be made with former examinations is a feature of great value as it affords an accurate method of determining the progress of the disease and the efficiency of treatment.

It is thus shown that the Röntgen method of examination is of great value in adding to the knowledge obtained by other methods of physical diagnosis. This is particularly true of the earlier tubercular lesions, especially the deep-seated peribronchial infiltrations and bronchial adinitis. In more advanced cases it adds an element of greater accuracy and detail in localizing the affected areas and corroborates the physical signs, while in all it forms a mechanical method of registering the observations which can be compared with others at a later date.

FIG. 7.

Tuberculous consolidation and cavity in left upper lobe. Infiltration and cavity with calcification in right upper lobe of lung. Heart in anteroposterior position.

FIG. 8.

Tuberculous infiltration and softening with cavities in both upper lobes. Anteroposterior position of heart.

Fig. 9.

Peribronchial scar-tissue after recovery of patient under sanitarium treatment

Fig. 10.

Localized non-tuberculous empyema at base of right lung

Fig. 11.

Symmetrical pulmonary congestion due to failing compensation from mitral stenosis.

Gynæcology and Obstetrics

THE EARLY DIAGNOSIS OF CANCER OF THE UTERUS; OPERATIVE TECHNIC *

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In the various portions of the mucous membrane of the uterus three definite varieties are to be found. The mucosa covering the vaginal portion of the cervix is made up of squamous epithelium; that between the external and internal os consists mainly of racemose or branching glands which secrete a varying quantity of mucus, whereas in the portion that lines the cavity of the uterus are numerous tubular glands differing totally from those of the cervix. From each of these types of mucosa cancer may develop.

In cancer of the uterus there occurs an outgrowth from the surface of the mucosa, while at the same time the growth penetrates into the underlying tissue. The cancerous tissue in the beginning is so rich in blood-vessels that the slightest disturbance of the growth is liable to cause bleeding, and not infrequently an increased blood pressure is sufficient to bring about a faint show. As the growth advances the older and more friable portions become necrotic and there results a breaking down which gives rise to a watery discharge often tinged with blood and frequently fetid. This discharge is usually the first symptom of cancer, but it may be totally wanting until the growth has reached large proportions. Cancer of the uterus is most common between the 35th and 50th years, but it is occasionally noted in patients under 25 years of age.

Any bloody or watery vaginal discharge that cannot be definitely accounted for demands an immediate and careful local examination.

* This paper forms part of a Symposium on the Early Diagnosis and Treatment of Cancer, arranged by the Pennsylvania State Faculty of Medicine. The various papers were read at the State Meeting in Philadelphia, September, 1909.

If on bimanual examination the cervix is found to be rough, friable and bleeding, the diagnosis of cancer is usually certain; but if the cervix is still intact the diagnosis may be very difficult. In early carcinoma of the cervix, when no disintegration has occurred, the surface is usually nodular and springing from it are fine finger-like outgrowths which bleed freely.

In every case a careful history should be taken but even after all possible data have been obtained and after a thorough bimanual examination, it will not rarely happen that the physician cannot determine to his satisfaction whether malignancy exists or not. In such cases a wedge of the suspicious area about 1 cm. deep and 2 or 3 mm. broad should be cut out, dropped at once into a 10 per cent. formalin solution and sent to the pathologist, who in the course of a few days will be able to decide with almost absolute certainty whether cancer is present or not.

When the cervix on bimanual examination appears normal the lesion is usually situated in the cervical canal or in the cavity of the uterus. The finding of an enlarged and nodular uterus renders it probable that myomata are present. When myomata are of the submucous variety the monthly periods are usually excessive, but as a rule no intermenstrual bleeding occurs and no fetid discharge exists, unless sloughing of a submucous nodule is going on. In such a case, however, portions of the growth are apt to be found projecting from the cervix, and such sloughing masses are readily distinguishable from cancer of the cervix, because they are tough and not friable, and also because the finger can be swept around them proving that they have originated at a point higher up.

Extra-uterine pregnancy frequently gives rise to intermenstrual bleeding and occasionally to a slight menstrual discharge, but in these cases we usually have a history of a missed period or of a period that has persisted and in addition there is frequently the localized pain caused by the gradual distention of the tube. Finally the bimanual examination will often reveal the definite velvety mass to one side of the uterus.

Pelvic inflammatory conditions are at times accompanied by a bloody or watery vaginal discharge. In these cases we can usually learn that there has been some recent local vaginal in-

fection or that an old pelvic lesion has recently been rekindled. In such cases there is often more or less elevation of temperature whereas in early cancer there is no fever.

When the patient is stout a satisfactory bimanual examination is often impossible, unless an anæsthetic is employed. When the cervix feels normal, and when we have excluded myomata of the uterus and lesions of the adnexa, the cause of the bleeding usually lies in the cervical canal or in the cavity of the uterus. Whatever the uterine growth it must drain into the uterine canal, otherwise there would be no vaginal discharge.

In such cases the uterus should be most thoroughly curetted. The mucosa should be brought away from the anterior, posterior and lateral walls and likewise from the cervical canal. If much tissue is obtained it is probable that malignancy exists. All of this tissue including the blood should be thrown into a 10 per cent. formalin solution without preliminary washing and sent to the pathologist.

In some cases the pathologist has considerable difficulty in saying whether a given specimen is cancerous or not, but as a rule there is just as much difference under the microscope between cancerous and healthy mucosa as there is between two totally different patterns of wall paper. Although there is always a possibility of error, so exact is the aid obtained from the microscope in the examination of scrapings that, in every instance during the last 16 years in which we have made a microscopic diagnosis of cancer, and have later examined the uterus, definite macroscopic evidence of cancer was present. Naturally a thorough knowledge of the various pictures due to faulty hardening of the tissues, to gland hypertrophy with or without pregnancy, and those peculiar to the normal mucosa, in early life, during menstruation and in old age, is necessary before one can undertake to pass judgment on the character of scrapings.

From no other part of the body is it possible to obtain material so easily for diagnosis. Take for instance cancer of the stomach. How thankful the operator would be were it possible to introduce a straight curette down to the pylorus and bring away some tissue for diagnosis, without the necessity of making any incision or of doing any suturing. For the early diagnosis of cancer of the

stomach an exploratory operation is necessary. We as general practitioners and gynæcologists have absolutely no excuse for failing to diagnose cancer of the uterus within one week after the first time the patient comes under our observation.

The Best Operative Technic.—To speak of the operative technic for cancer of the uterus before a Pennsylvania audience is akin to bringing coals to Newcastle, when among others of your number an old friend of mine, Dr. John Clark, has contributed so much to our knowledge of the subject. The operation as elaborated by Wertheim seems to offer the best results. It consists in the removal of the uterus, appendages and parametrium and often also of the pelvic lymph-glands.

It is now generally recognized that the greatest dangers of the radical operation are due to the shock that immediately follows. More recent experience has shown that it is possible to lessen this shock in two ways: (1) by shortening the duration of the operation, (2) by keeping the patient warm while she is on the table and afterwards.

With the abundant flood of "sunshine" as furnished by Krönig's light the operator can often save from fifteen to thirty minutes. This light should be in every operating room where much abdominal work is done.

The chief bleeding encountered in the radical operation is from the vaginal plexus of veins. The long, short-curved Wertheim forceps enable one to clamp and cut the vessels with great facility, thereby often saving from ten to fifteen minutes. As was said before, this shortening of the operation by even ten minutes is all important.

By means of Krönig's electrically heated table the patient can be kept at an equable temperature and usually leaves the operating room in an infinitely better condition than when the ordinary table is used. The patient is cleaned up on the regular table, and after being thoroughly dried is placed on double blankets laid over the electric table. It is always necessary to have a special nurse or assistant guard against any danger of burning the patient, and the current should be turned on and off as necessary in order that a reasonably equable temperature may be maintained. In the course of a few weeks I operated on six cancer

patients in succession and although in some of them the operation was a most extensive one and the patients were weak, in not one case did there occur the marked degree of postoperative shock so often noted.

In the time at my disposal I have merely sketched the salient features in the diagnosis and treatment of cancer of the uterus and have omitted any consideration of polypi, adenomyoma, sarcoma and chorio-epithelioma.

In conclusion I cannot refrain from quoting an appeal made by the late J. Knowsley Thornton some years ago—at once an appeal to us as medical men and one of the severest arraignments of our profession that has ever been made in our management of cancer of the uterus.

"How is an early diagnosis to be made? Clearly by neglecting no menstrual departure from the normal, however trivial it may at first sight appear, but at once to encourage the patient to accurately describe symptoms, and above all to insist in the most determined manner on a local examination. Here it will be apparent that I, as a consultant, appeal for help to the great body of those who are now listening to my remarks, to my professional brethren engaged in general practice. I, in common with those situated as I am, too seldom have an opportunity of diagnosing early, because the majority of the patients come to us too late, when the disease has already advanced nearly, or quite, beyond the limits of surgical aid. Let me then appeal to all engaged in family practice who listen to me here, and to that larger body who may read my words when reproduced in the medical journals, to sternly cast aside that too great modesty, or that tendency to treat as trivial, small symptoms, and to at once take alarm about, and carefully investigate, every case in which there is brought to their notice an abnormality in menstruation, or a vaginal discharge of any kind, however trifling. A very grave responsibility lies at the doors of the medical profession for the small progress made in the early diagnosis of uterine cancer and its successful treatment. How constantly is the consultant told: 'I mentioned it to my doctor weeks or months ago, but he said, "Oh, it is nothing; I will send you a little medicine or a little injection," and never even suggested any internal exami-

nation, so I did not like to trouble him again till the pain became so bad or the discharge so troublesome, and then he examined me and said I must have special advice at once.' Invaluable weeks or months gone, and then the verdict of the consultant, 'It is not a case for operation,' which really means 'You have come too late,' but cannot be so candidly expressed, because he must guard the reputation of his professional brother. I admit that the false modesty of the patient, especially in some classes of society, makes the position a difficult one, especially for the young family doctor, but let me implore you all to awake to what is at stake, and to be firm in your demand for an examination, and if you have any doubt after such an examination, to urge that the patient should at once seek the advice of some one who has larger opportunities than yourself for forming a sound opinion. I will go one step further, and ask you, if there should be any to whom such a temptation comes, never to go on treating a case in which there is a shadow of doubt, either because you doubt or because you want practice; if the case is susceptible of treatment at all, it is only surgical treatment which can avail, and that of so severe a kind that it requires the knowledge of the specialist if ever any disease did or does require special knowledge and special skill in operative treatment."

GRAVITY METHOD OF REMOVING ADHERENT PLACENTA

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The frequency with which I have been called to post-parturitive complications, superinduced by residuary portions of adherent placenta which had not yielded to the customary force methods employed by practitioners or midwives, together with a number of cases of stubborn adherence found in my own practice, convince me that some other method of removal more reliable than the procedures commonly followed should be devised. It is obvious that my conviction led to careful study and experiments, ultimately producing results which I feel will prove very interesting to my colleagues.

A very short period of practice suffices to bring the physician into contact with many cases where wasting after parturition has continued for weeks and months to the physical discomfort and systemic detriment of the nea mater. Such symptoms naturally indicate the necessity of curettement as the first practical resort.

In such cases it is common to find a purulent discharge flowing from the uterus and every evidence that the bacterial invasion has insidiously traversed the Fallopians and irreparably damaged those highly important and exceedingly sensitive organs, the ovaries.

The cause of such inflammatory condition is directly traceable, either to lacerations of the uterus, or, more frequently, to incomplete removal of the placenta and probable imperfect performance or entire neglect of necessary curettement.

Where Nature in very ordinary cases expels the secondi in normal manner, the possibility of a residuum in the uterus, always exists. It is therefore manifest that in adherent placenta the possibility of such residuum is multiplied, especially where the heroic method of manual force is employed for the removal of the structure.

Objections to the usual force methods may be justly summed up as follows:

1. Inherent risk of infection owing to the introduction of the hand into the uterus.

2. Extreme liability of uterine lacerations by reason of an error of judgment as to the intensity and direction of manual force to be exerted.

3. Imminent probability of an incomplete removal of the placenta.

4. Frequency of death owing to hasty removal of adherent placenta by force and attendant complications.

Digressing somewhat from the subject in hand, I consider it opportune to state here that during the past year the territory in which I am active seemed to be extraordinarily redundant of post-parturitive complications and other curious abnormalities in connection with my obstetrical practice; yet, I did not attend more accouchements than in other years. Two children without eyes were delivered, each having a dark cavity at the root of nose as though containing coroid or retina, but vitreous lens and cornea missing. Both children were still-born and had what resembled a double head with a constriction between the two formations. I also had one face presentation and one prolapse of the cord. Two cases of placenta previa and one of eclampsia likewise occurred within the same period. I also found streptococcus extraordinarily virulent and I experienced more difficulty in combatting infections than in all the previous fourteen years of my practice taken collectively. In addition to the above I had two cases of adherent placenta which will later illustrate in this article the method which I think will commend itself as a logical deduction from the premises presented.

During the past five years two cases where the attendant physicians removed adherent placenta hastily and by force, came to my especial notice. Curettement had been either neglected entirely or imperfectly performed and as a consequence the women in question were left in a deplorable condition. Bleeding had continued during seven and eight months respectively and ovarian disturbances caused continual side-ache, and backache, weakness and exhaustion, with the distraught features and pale mucous membranes reflecting a picture of suffering and woe which could not fail to excite sympathy. And yet, even though in such physical torment, these mothers must nourish their infants and discharge

the household drudgeries which *per se* are without alternative to the wives of laborers whose battle for existence yields nothing in excess of bare necessities.

There are few physicians who have not heard of the death of a parturient woman resulting either directly or indirectly from forcible removal of adherent secundi. I know of ten deaths among parturient patients during the past year. Of these deaths eight were caused by sepsis superinduced by various conditions, while two were due to rupture of the uterus in removing adherent placenta by manual force.

The report of these two deaths in addition to the many experiences I had had in personally ministering to patients suffering from the effects of residual portions of forcibly removed adherent placenta impelled me to seek some way by which I might successfully meet in my own practice such parturitions as might develop secundi of similar character.

It is obvious that I was fully convinced that whatever method was to be devised must of necessity be free from the positive risks known to be concomitant with the customary procedures. The evolution of my solution of the question is given in the following train of reasoning. None of the text-books extant contain any reference to a similar method nor have I ever heard of any similar proposition.

It seemed reasonable to presuppose that if—as had been frequently proven—a portion or portions of the placenta could remain in the uterus for weeks and months without directly incapacitating a woman, then the entire placenta might remain for a short period without incurring any greater risk.

Furthermore under normal conditions Nature shows every disposition to cast off the placenta after its function has been completed, a circumstance of vital import in the consideration of ways and means to be employed in removing the structure when adherent.

The severing of the umbilical cord and resultant stoppage of circulation must necessarily have a distinct effect on the placenta. Such metamorphosis would more likely tend toward the eventual expulsion of the placenta than toward increasing its adherency.

I decided therefore in any case where the placenta proved adherent to combine patience and gravity and refrain from the customary speed and force.

The following cases will illustrate the application of my conclusions.

Mrs. F. B., age 25, primipara, had been in labor five (5) hours, child being born without requiring the use of forceps. No difficulties presented themselves until placenta was to be delivered. Crede's method was of no avail; and the combination of this method with a slight traction on cord also proved unsuccessful. I spent over an hour manipulating the uterus but the placenta would not respond. After careful disinfection of the hands I examined the interior of the uterus only to find that the placenta was so closely adherent as to make it seem a portion of the womb.

To have attempted to separate the placenta from the uterus by usual methods would certainly have meant rupture or laceration of the walls of the uterus, probable intra-uterine hemorrhage and possibly a fatal termination.

The time seemed especially opportune for an application of the theory of patience and gravity. Accordingly I tied a piece of gauze bandage to the cord and attached to the free end of the bandage half-a-brick wrapped in a white cloth. The brick was so adjusted as to permit it to hang over the foot of the mattress at a height of about 16 inches from the floor. Visiting her daily I found that her temperature remained nearly normal, at no time rising above 99°. The cord became dried, smooth and shiny, resembling greatly the string of a bass viol. On the fourth day when I arrived I was informed that the brick had "fallen," much to the surprise of the young mother as well as to the women of the neighborhood whose curiosity to learn what the "brick was for" prompted their making frequent visits. The placenta was delivered intact, Mrs. B. pronouncing the pain she had experienced during the four days as rather trivial. A slight laceration of the perineum, which had been sewed with chromicized catgut, healed perfectly.

On the tenth day Mrs. B. left her bed in splendid condition and has not required any medical attention since.

The second case was Mrs. M. B., age 34, mother of three children. She had had an abortion of 3 months. She suffered much loss of blood and severe pain during three months and finally consulted me in her efforts to secure relief. I curetted away a number of pieces of adherent placenta, after which her condition

became much improved. The right ovary however was enlarged and congested. Becoming pregnant later Mrs. M. B. anticipated much trouble and suffering during her impending confinement. When I was called to her bedside I found the child in the L. O. A. position with the fetal heart sounds regular and labor progressing nicely. In three and one-half hours the child was born without recourse to forceps. I waited fifteen minutes and then tried to express the placenta by the Crede method, but without effect. I then made traction on the cord as much as allowable without incurring risk of rupturing same from the placenta, manipulating the uterus through the abdominal wall all the while. My efforts, however, were without success.

After waiting an hour I finally decided again to use the gravity method. After having attached the half-brick to the cord via the gauze bandage as explained in the foregoing case, I advised Mrs. M. B. to feel contented that even though the method I was using was new to her and would probably appear so curiously novel to her neighbors as to evoke criticism, I nevertheless felt positive that the adherent placenta would be removed in this manner with very little inconvenience to her. On the afternoon of the fourth day the brick dropped to the floor much to the satisfaction of the mother and myself. Her temperature at no time rose 2 degrees above normal. She stated that the presence in uterus of the placenta caused her slight inconvenience but she was perfectly satisfied to endure a little pain for a few days rather than suffer forcible removal. Mrs. M. B. arose on the tenth day. For the right ovary affected by previous miscarriage I gave her some Ichthyol suppositories. She asserts that the ovary does not cause her any pain and has enjoyed good health since the confinement in question.

In both the above cases I gave small doses of fluid extract of ergot, 10 min. three times a day, in order to prevent relaxation of the uterus and probable intra-uterine hemorrhage.

Both women were very grateful at the ease with which they were relieved, assuring me that they had suffered no unusual pain or marked inconvenince during this slow but certain method.

From the above it would seem to me that the method described is a safe one. With small doses of ergot three times per day, and a douche of 1 dram Lysol to 2 quarts of water once or twice a day, so long as no sepsis is manifested, the practitioner may

depend on the removal of the adherent placenta by the gravity method.

The weight to be used for the tractile purpose must naturally be selected with discretion so that no uterine laceration or eversion results.

I also advise placing under the castors at the foot of the bed a block or brick so as to elevate the foot of bed from two to four inches. The slope thus created lessens the likelihood of the patient's slipping toward the foot of the bed. Furthermore it allows a little more fredom of motion, because the weight has more travel before it could reach the floor in which position its tractile effect would be null.

I think the method will commend itself particularly when we consider the possibility of finding adherent placenta after protracted and severe labor which has left the woman exhausted. Under such circumstances it is desirable to reduce further suffering as much as possible. If adherent placenta is removed then by force, the shock to the parturient woman is liable to sap her recuperative powers to an extent difficult to surmount. Should such a case be further complicated by intra-uterine hemorrhage as a result of the forcible removal of the placenta the danger of a fatal termination would certainly be materially increased.

In conclusion it is interesting to glance at statistical office-records and ponder over the great number of deaths recorded as having been incident to parturition. Deaths of this kind are entirely too frequent under conditions which are so near normal that they should have insured prompt and perfect recovery. Such deaths would seem absolutely contradictory to Nature's provisions for the propagation of species.

Where abnormalities complicate matters the liability of death is of course naturally increased. Again, methods which are our only alternative in treating with such abnormalities involve risks of greater or less importance. Some of these risks are collective in that they not only expose the patient to death, but in the event of survival mean, almost to a degree of certainty, the impairment of health.

If then my exploitations in reference to adherent placenta shall have in any measure assisted in reducing the mortality common in such cases even in a small degree I shall be very happy.

OVARIAN FIBROMATA

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Considered from the viewpoint of either etiology, pathology, or histology, ovarian fibromata differ in no essential respect from fibroid tumors encountered in other situations. According to a statement accredited to Bland-Sutton, pure ovarian fibromata are so exceedingly rare that when observed they may be regarded simply as "pathological curiosities." Moreover, current surgical textbooks scarcely mention the possible occurrence of fibromatous neoplasms involving the ovaries or Fallopian tubes; Nicholas Senn (Pathology of Tumors) admits having observed, in his extensive surgical experience, but two instances of pure ovarian fibromata; and Taylor (*Edinburgh Medical Journal*) says that fibroid tumors of the ovary (even co-existing with fibromyomata of the uterus) are extremely rare, and when they occur there is always a "decided tendency to torsion of the pedicle," a statement which is hardly borne out by facts, since twisted pedicle appears to be no more frequent in fibroma than in other pedunculated ovarian tumors.

Like other neoplasms of ovarian origin, fibromata may be either unilateral or bilateral. It is exceptional, however, for both ovaries to be involved, and in any event the Fallopian tubes usually remain free from disease. Pain and discomfort, the result of pressure upon and interference with the functions of other important structures, are usually prominent features; and ascites is not infrequently a concomitant symptom. In not a few instances the latter complication has induced the patients to seek surgical relief; indeed, ascites, in the absence of definite indications of malignant disease involving the pelvic or abdominal viscera, should remind one of ovarian fibroma as the possible causative factor (Senn). While pain is commonly present throughout the development of all ovarian fibromata, examples have been recorded where this symptom was entirely absent.

It has been shown that fibroid tumors of the ovary occur with

greater frequency in women between twenty and thirty, yet such neoplasms have been observed in those much younger, and also many years older, *e.g.,* one author (Leopold) cites thirteen instances in patients from five to thirty, only four between thirty and forty, and Dauber removed a large ovarian fibroma from a woman seventy-seven years old.

Differential diagnosis, based upon physical examination, between ovarian fibroma and other solid tumors involving the ovary and Fallopian tube may be exceedingly difficult; and between fibroid of the ovary and pedunculated uterine fibromyoma differentiation is ordinarily quite impossible except by exploratory abdominal section. As a matter of fact, a correct ante-operative diagnosis of ovarian fibroma is seldom practicable, for reasons too obvious to require enumeration; but since the treatment would be uninfluenced in any event, the question of accuracy in diagnosis is not of paramount importance.

An interesting example of fibroma of the Fallopian tubes is recorded by Dunning (International Clinics) in a woman of twenty-six, who had been married five years. The patient had suffered from (so-called) "recurrent diffuse pelvic inflammation" since abortion about four months after marriage. Provisional diagnosis of bilateral pyosalpinx with small ovarian cystomata was made, and proved partially correct at abdominal section since both tubes contained pus. A fibroid nodule occupied or completely surrounded the uterine extremity of each Fallopian tube, almost totally occluding the lumen. In commenting on the case the author observed: "We have here a rare pathological specimen, the first of the kind that I have ever found. Fibromata of the Fallopian tubes are rarely seen. Rokitansky mentions them, and Simpson describes a case in which the tumor was as large as a child's head. They are usually subserous, but may be interstitial (according to Winckel), and of such a character is ours." Microscopical examination showed nodules composed of unmistakable non-inflammatory fibrous tissue. Of course this case is not cited as one of ovarian fibroma, yet the rarity of fibromatous neoplasms involving either the Fallopian tubes or ovaries seems to amply warrant interpolation of the abstract in this connection.

Careful and painstaking search of available surgical literature

from about 1840 to date brings to light record of less than one hundred examples of true ovarian fibromata. In the series of cases, abstracts of which are herewith presented, the writer has excluded all those in which there appeared any reasonable doubt as to correctness of diagnosis or classification after careful consideration of all the pathological and microscopical data given. And any one who has ever attempted to perfect a compilation of this scope and character, based as it must necessarily be largely upon published reports, will readily understand and appreciate the difficulties encountered in the task, the amount of labor required in examining, excerpting and preparing the records, and the care which of necessity must be exercised to guard against palpable error in presentation of the accumulated statistical data. Furthermore, the pertinent fact must not be permitted to pass unobserved that medical and surgical records are not always strictly trustworthy, and regardless of the care used in the selection and classification of the material presented, the writer cannot be expected under the circumstances to vouch for its absolute correctness in every detail, *e.g.*, owing to faulty microscopical technic on the part of the examiner mistakes in classification are not impossible, and if such be true, detection and correction by the compiler of the statistics would naturally be impracticable.

EXHIBITS

No. 1 (*Wilks*).—The author exhibited specimen of ovarian tumor weighing three pounds from a woman aged fifty-seven. The growth occupied left side of abdomen reaching to hypochondrium, duration unknown, originated in left ovary, long pedicle, smooth, oval, no adhesions. Tumor exceedingly hard, incised with difficulty, composed of fibres interlacing and curving in various directions, showing concentric circles. In midst of tumor small vascular mass softer than surrounding parts, probably remains of left ovary, although no characteristic ovarian structure was detected. Microscopical examination showed tumor composed of simple white fibres (mixed with some nucleated fibres), numerous long, flattened bands indistinguishable from (so-called) muscular fibres of uterine fibroma. Postoperative diagnosis—ovarian fibroma.

No. 2 (*Ibid.*).—Specimen from woman aged fifty-four, abdominal tumor ten years, last labor retarded thereby. Growth weighed

three pounds, involved right ovary, attached to uterus merely by broad ligament. Hard, smooth, oval; structure uniform throughout, simple fibres having few nuclei, no distinct fibrous bands. Pure ovarian fibroma.

No. 3 (*Wells*).—Patient aged sixty-four succumbed to cardiac and renal disease. Physical examination during life demonstrated tumor lying behind bladder, depressing anterior vaginal wall, pushing uterus backward. Its presence had occasioned little suffering, surgery not invoked. Postmortem—tumor size of cocoanut in left ovary, so hard that saw necessary for division, uterus and right ovary normal. Microscopical examination showed tumor composed of fibrous tissue, denser parts calcified by deposition of carbonate of lime. Post-mortem diagnosis—true ovarian fibroma, calcareous degeneration.

No. 4 (*Ingham*).—Patient aged twenty-nine, married, five children, last two born at seventh month. Abdominal pain and general ill-health since birth of last child, five years previously. Tumor size of orange above pubes, movable, slightly painful, rapid enlargement, extensive ascites, fourteen quarts fluid liberated. Physical examination showed hard, non-fluctuating, immovable growth in right pelvis extending three inches above pubes posterior to uterus. Condition of patient precluded surgical intervention. Paracentesis practised many times later, death from peritonitis. Necropsy revealed pelvic tumor occupying so closely superior strait that great difficulty was experienced passing knife far enough to extract entire pelvic viscera; uterus normal. Left ovary contained cyst size of marble, right ovary had disappeared, its place was occupied by solid tumor size of adult head, adhesions extensive. Post-mortem diagnosis—ovarian fibroma.

No. 5 (*Goodhart*).—The author exhibited specimen of ovarian fibroma secured post mortem from woman aged forty-two who died as result of granular kidney disease. Tumor originated in free (epithelial) surface of ovary, had no connection with broad ligament. Section showed it to spring from outer layer of ovarian structure, that part characterized by older writers as "tunica albuginea," and as the condensed layer of proper ovarian stroma by Waldeyer. Growth one and a half inches in diameter. There were also several fibrous tumors in uterine wall.

No. 6 (*Walsham*).—The author presented specimen of pure ovarian fibroma obtained from dissecting room subject. Growth size of orange and described as having its origin in fibrous capsule of left ovary.

No. 7 (*Virchow*).—The author describes a tumor which originated in free end of right ovary, 5.5 cm. in length and breadth, and 4 cm. in thickness, attached to which were the ovarian ligament, the intact portion of ovary with thickened tunica albuginea, and several corpora fibrosa. Microscopical examination showed no definite muscle fibres, thick connective tissue formation, small spindle and recticular cells, and some portions that had undergone fatty degeneration.

No. 8 (*Leopold*).—The author describes an example of typical fibroma of the ovary size of walnut, and states that no trace of ovarian tissue could be detected in specimen.

No. 9 (*Crisp*).—Patient aged fifty, unmarried. Physical examination—large, firm tumor in lower abdomen, presumably connected with uterus. Patient exceedingly feeble, abdominal pain, vomiting, often dark colored coffee-like material, surgery not invoked, death eight days later. Postmortem showed solid ovarian tumor weighing eight pounds, globular, nodulated, attached by small pedicle to left ovary, composed throughout of dense fibrous structure, some portions congested. Post-mortem diagnosis—ovarian fibroma.

No. 10 (*Cullingsworth*).—Patient aged thirty-six, married, five children, last one five years previously, uterine hemorrhage three months. Physical examination showed two hard, nodulated pelvic tumors, the right large as man's fist extending in front of uterus, freely movable; larger left tumor behind uterus extending upward to fundus, slightly movable on manipulation, no operation. Death forty-three days later. Autopsy—right ovarian tumor in front of uterus and right broad ligament, weight 255 grammes; left ovarian tumor wedged in behind uterus, weight 624 grammes; both solid throughout (except serous cyst one inch diameter in right growth), nodulated, whitish-gray in color with smooth glistening surface. Microscopical examination showed fine and coarse fibres, cellular elements, blood-vessels, microscopical cysts; some cells arranged in vertical or transverse rows and concentric circles, pos-

sessing all characteristics of tissue corpuscles, oval nucleus, long cell body, fine outrunners, separated from each other by clear intercellular fluid, and fine fibrillæ. Post-mortem diagnosis—ovarian fibromata.

No. 11 (*Powpow*).—Patient aged thirty-eight succumbed to cirrhosis of liver and ascites. Necropsy revealed tumor of right ovary size of small cocoanut, left ovary large as hen's egg. Microscopical examination showed small peritoneal knots of lymphomatous structure with new growths of connective tissue originating in ovarian stroma; both tumors irregular, solid in consistence. Tissue surrounding the myomatous structures, particularly near vessels, consisted of numerous round elements between which numbers of transition forms were observed. Instead of follicles in different stages of development, tubular and sac like structures found in connective tissue, representing the intercellular spaces and lymphatic channels filled with cell elements. Thus entire tissue of ovary, excepting vessels, consisted of numerous compact portions of connective tissue intermingled with myxoma, and lymphatic spaces whose cell elements had undergone mucoid degeneration. Post-mortem diagnosis—double ovarian fibromyxoma.

No. 12 (*Williams*).—Patient aged eighteen, hard, painless, movable abdominal swelling for two years, rapid increase for last six weeks, growth reached ensiform cartilage, uterus normal and movable. Menstruation appeared at seventeen, regular, but missed period before operation. Abdominal section—right ovarian tumor, eight inches longest diameter, firm to touch, soft at certain points, with long thin pedicle. Postoperative diagnosis—calcifying ovarian fibroma, cystic and calcareous degeneration.

No. 13 (*Ibid.*).—Patient aged twenty-seven, single, menses regular. Hard, painful, irregular abdominal tumor on right side for two years, gradual development. Abdominal section—right ovarian tumor, twisted pedicle, adhesions to abdominal parietes, cæcum and colon. Lobulated and nodular ovarian fibroma, hemorrhagic area in interior, ovarian ligament attached to hilum.

No. 14 (*Polk*).—The author exhibited two ovaries, secured post mortem, which were the seat of marked fibroid degeneration. Patient a nursing woman. Physical examination during life revealed two abdominal tumors supposed to be uterine fibromata with long pedicles. The aid of surgery not invoked. Patient suc-

cumbed fourteen days later from rapidly ascending paralysis. Necropsy showed double ovarian fibroma, tumors large, apparently interstitial in character.

No. 15 (*Ibid.*).—The same author presented another ovarian tumor size of gravid uterus at fifth month. Patient aged twenty, abdominal growth noted several years, slow development, opposite ovary cystic, double oöphorectomy. Postoperative diagnosis—ovarian fibroma.

No. 16 (*Doran*).—The author exhibited (London Pathological Society) small spherical tumor which had its situs in the substance of left ovarian ligament. Microscopical examination showed tumor composed of plain muscular fibres mingled with white fibrous tissue. Patient also had small uterine fibroid for which abdominal section was performed. Postoperative diagnosis—fibromyoma of ovarian ligament.

No. 17 (*Carter*).—Patient aged twenty, single, abdominal tumor six months, girth increased one inch last few weeks. Amenorrhœa three months, menses regular thereafter. Abdominal section—right ovarian tumor, smooth, very hard, weight ten and three-fourth pounds. Postoperative diagnosis—fibromyoma of right ovary.

No. 18 (*Williams*).—The author exhibited ovarian tumor consisting of two parts, a lower solid and upper cystic, the whole weighing seven and a half pounds. Solid portion spheroidal, flattened anteroposteriorly, covered by smooth mottled membrane through which distended vessels were visible, surface raised in several places into small distended bullæ containing dark grumous fluid, substance of tumor softer under these than elsewhere. At lower portion of growth nodule size of hen's egg. Section is reddish-gray, tough fibrous tissue, in centre numerous spaces having smooth surface filled with bloody fluid. Attached to upper posterior of surface of solid growth is large collapsed cyst with thin, tough walls, which close to pedicle become thicker. Inner surface is smooth and glistening, near thickened portion of wall studded by reddish papillomatous growths varying from pin head to hazelnut, in largest of which cyst size of pea observed. Pedicle thick, twisted, containing extravasated blood and large veins filled with coagulum. Solid tumor ovarian fibroma.

No. 19 (*Bantock*).—Patient aged forty-seven, widow, two chil-

dren, last confinement twenty-three years previously. Tumor size of hen's egg in right groin one year, rapid development. Physical examination—prominent heart shaped, firm, movable enlargement in hypogastrium. Abdominal section—right ovarian tumor, omentum extensively adherent, pedicle traced to right side of uterus, left ovary atrophied and corrugated. Microscopical examination—principal portion of growth dense, white, fibrous tissue containing numerous rounded various sized bodies, each having capsule represented by glistening, wavy band. Underneath these bands undefined granular looking material is closely attached to capsule, believed to be Graafian follicles destroyed by pressure from surrounding fibrous tissue. Postoperative diagnosis—ovarian fibroma.

No. 20 (*Schachner*).—Patient aged thirty-five, abdominal enlargement several years duration accompanied by profuse menstruation. Physical examination showed immovable tumor in median line extending above umbilicus, cervix drawn upward and firmly fixed. Exploratory laparotomy—tumor weighing fourteen pounds, originated in right ovary, hard, nodulated, dense adhesions, omental vessels greatly enlarged. Postoperative diagnosis—ovarian fibroma.

No. 21 (*Ibid.*).—Patient aged fifty-five, hard, movable tumor noted in right side of abdomen several years. Abdominal section—ovarian fibroma weighing four and a half pounds, long pedicle.

No. 22 (*Senn*).—Abdominal tumor recognized ten years before its removal was permitted, woman insisting upon repeated postponement of surgical intervention, only agreeing thereto in hope of obtaining relief from ascites. Abdominal section—ovarian tumor weighing twenty pounds, firm in consistence, pedicle slender, no adhesions. Microscopical examination—wavy bundles fibrous tissue interlaced in all directions, blood-vessels few and small. Postoperative diagnosis—ovarian fibroma.

No. 23 (*Ibid.*).—Patient had pelvic tumor several years. Abdominal section—ovarian tumor weighing ten pounds, presenting characteristics not dissimilar from foregoing example. Discomfort due to ascites induced patient to seek surgical relief.*

* The author remarks that in both instances cited the peritoneum was exceedingly vascular, which he believed due to its having been "thrashed by the tumor" for a number of years [M.].

No. 24 (*Feiss*).—The author describes right ovarian tumor size of goose egg, shape of normal ovary, surrounded by firm capsule easily stripped off except few spots firmly adherent. Capsule of firm connective tissue poor in vessels, below this a layer of looser connective tissue with strands widely separated and rich in vessels. The growth had so stretched ovary on its surface that capsule was formed from the tunica albuginea and compressed ovarian stroma. Tumor ovarian fibroma.

No. 25 (*Crawford*).—The author exhibited specimen from woman aged forty-nine who committed suicide. Married at eighteen, two children within three years. Soon after birth of last child severe hemorrhage followed operation on uterus, nature unknown. Postmortem—tumor left ovary size of orange, weight six and a quarter ounces, hard, irregularly globular, covered by peritoneum, small patch mucoid degeneration one side. Section—solid throughout, the ovary from the hilum of which growth apparently arose was spread over tumor. Microscopical examination—pure ovarian fibroma.

No. 26 (*Horrocks*).—The author presented specimen removed from girl aged twenty-two. Pelvic tumor displacing uterus upward and forward, removed by abdominal section. Growth originated in left ovary, ovoid shape, twelve inches in circumference, surface smooth, non-vascular, hard, when incised remained flat like cut surface of raw potato, only watery fluid obtained on scraping. Microscopical examination—tumor rich in cellular elements, cells having characteristics of emybryonic tissue, intercellular stroma in some places showed distinct white wavy fibres. Pure ovarian fibroma.

No. 27 (*Baldwin*).—Patient aged fifty-five, married, five children, no miscarriages, menopause ten years previously. Severe pelvic pain several weeks duration, general condition wretched. Physical examination—abdominal tumor extended to umbilicus. Abdominal section—solid ovarian tumor weighing four and a half pounds, long pedicle three times twisted. Postoperative diagnosis—ovarian fibroma, extensive calcareous degeneration.

No. 28 (*Purslow*).—Patient aged twenty-six, unmarried. Abdominal pain, swelling of several months duration, menstruation scanty, defecation painful, frequent micturition. Physical exam-

ination—abdomen considerably distended; firm, nodular, irregular tumor right side reaching to umbilicus; ascites. Abdominal section—three pints ascitic fluid, tumor right ovary, weight three pounds, solid throughout, composed entirely of fibrous tissue some of which had undergone fatty degeneration. Typical ovarian fibroma.

No. 29 (*Doran*).—Patient aged twenty-four, single, one year previously suffered sudden attack retention of urine, relieved by catheter, abdominal tumor five months. Physical examination—movable tumor, stony hardness, extending to umbilicus median line, irregular process reached much higher left side, hymen intact, uterus behind growth. Abdominal section—ovarian tumor right side, pedicle twisted, ovarian ligament hypertrophied, pint red serum in pelvis, no adhesions, opposite ovary uninvolved. Irregular, hard, tumor, weighing three and a half pounds, small cyst on surface. Microscopical examination showed usual characteristics of ovarian fibroma, few free connective tissue elements.

No. 30 (*Ibid.*).—Patient aged fifty-two, lump in right iliac fossa eighteen months, slow development. Physical examination—hard mass in hypogastrium, uterus quite separate. Abdominal section—right ovarian tumor, broad pedicle consisting of Fallopian tube and mesosalpinx, ovarian ligament hypertrophied, several ounces clear serum in Douglas's pouch, infundibulopelvic ligament œdematous, left ovary atrophied. Very hard, smooth tumor, weight three pounds, typical ovarian fibroma, degenerated tissue in interior. Microscopical examination—dense stroma pure fibrous tissue with distinct areas of young connective tissue.

No. 31 (*Briggs*).—Patient aged twenty-nine, abdominal tumor and pain one year, pregnant four months. Physical examination—stony hard tumor independent of uterus. Abdominal section showed left ovarian tumor size of small cocoanut, right ovary normal. Growth almost structureless—calcified ovarian fibroma.

No. 32 (*Ibid.*).—Patient aged thirty, abdominal tumor accompanied by pain on exertion, irritable bladder, duration one year. Physical examination—uterus normal, solid tumor in pelvis independent of uterus, ascites. Abdominal section—left ovarian growth size of orange, right ovary normal. Spindle celled ovarian fibroma.

No. 33 (*Ibid.*).—Patient aged thirty-two, painful abdominal

tumor four months duration. Physical examination—uterus small, solid independent tumor, ascites. Abdominal section—solid growth right ovary size of orange, left ovary normal. Spindle-celled ovarian fibroma.

No. 34 (*Ibid.*).—Patient aged sixty-eight, painless abdominal tumor three months, rapid development. Abdominal section—tumor right side, uterus normal, cervix elevated. Growth weighed seven and quarter pounds, adherent to sigmoid flexure, cæcum, appendix and pelvic peritoneum. Ovarian fibroma with spindle cells.

No. 35 (*Ibid.*).—Patient aged thirty, painless abdominal tumor eighteen months, some soreness, rapid development, acites. Physical examination—solid tumor independent of uterus, latter normal. Abdominal section—heavy, solid growth left ovary size of adult head, right ovary normal. Spindle-celled ovarian fibroma.

No. 36 (*Ibid.*).—Patient aged forty-eight, menopause six years previously, no symptoms except enlargement of abdomen noted fourteen days before operation. Physical examination—uterus normal, some ascites, tumor detected each side of abdomen. Abdominal section—double oöphorectomy, large tumor size of adult head, growth opposite side size of orange, three pints ascitic fluid. Double ovarian myxofibroma.

No. 37 (*Ibid.*).—Patient aged twenty-two, menstrual history normal until last two months scanty or wanting, painless abdominal tumor three months, bladder slightly irritable. Physical examination—large growth independent of uterus, ascites. Abdominal section—solid tumor right ovary size of adult head, several mucoid patches, left ovary normal. Cellular ovarian fibroma, mucoid degeneration.

No. 38 (*Ibid.*).—Patient aged fifty-five, menopause two years previously. Painless abdominal tumor size of cocoanut, rapid development last two years, ascites, uterus normal. Abdominal section—tumor originated in left ovary, solid except one superficial cyst, broad thick pedicle, right ovary normal. Ovarian fibromyoma.

No. 39 (*Knowsley*).—Patient aged twenty, married, no children, menstruation regular, abdominal tumor several months. Abdominal section—pure fibroma left ovary, right ovary uninvolved.

No. 40 (*Jones*).—Patient aged twenty-one, married, no children. Lump in right iliac region eighteen months, tumor reached above umbilicus, stony hardness, menses regular, no pain. Abdominal section—right ovarian tumor, long pedicle, pint clear fluid in peritoneal cavity, no adhesions. Stony hard tumor principally fibrous tissue. Typical ovarian fibroma.

No. 41 (*Dakin*).—Patient aged thirty-six, married, abdominal tumor, irregular, movable, one year; rapid increase in size last nine months, growth felt like cyst, menses regular. Abdominal section—right ovarian solid tumor weighing nine and half pounds, extensive omental adhesions, left ovary normal. Postoperative diagnosis—ovarian fibroma, some mixed spindle and round cells.

No. 42 (*Cullingsworth*).—Patient aged twenty-one, single, abdominal tumor two years, caused little inconvenience beyond slight attacks of pain, menstruation unaffected. Abdominal section—left ovarian tumor, oblong, solid, weight five and a half pounds, lower portion moulded to shape of pelvic cavity, upper part larger in all dimensions, extended above umbilicus. Growth round and smooth anteriorly, flattened posteriorly, short pedicle, few vascular posterior adhesions. Commencing cystic degeneration of intrapelvic portion, remainder, on section, presented appearance of œdematous fibrous tissue. Microscopical examination—simple fibroma, cystic degeneration.

No. 43 (*Doran*).—Patient aged forty-nine, married, tumor in hypogastrium, recently increased rapidly in size. Physical examination—extremely hard pelvic mass, immovable, as was also the uterus. Abdominal section—right ovarian tumor, pedicle short but five inches broad, three planes, layer of peritoneum forming special ovarian mesentery, mesosalpinx much elongated as was also ovarian ligament. Tumor weighed four and a half pounds. Pure fibroma with cavity in centre from degenerative changes.

No. 44 (*Morely*).—Patient aged twenty-two, single, small tumor right side two years. Abdominal section—right ovarian tumor size of small cocoanut, left ovary which showed fibroid degeneration also removed. Microscopical and histological examination showed small cysts, œdematous tissue, simple fibroma.

No. 45 (*Laidley*).—Patient aged twenty-nine, married, healthy child two months old. Hard, slightly movable abdominal tumor

size of cocoanut for two years, which, during pregnancy, was pressed upward to left side umbilicus. Physical examination—dense immovable growth left umbilical region, ascites. Abdominal section—ovarian tumor weighing two pounds. Histological report—ovarian origin shown by remnants of cortical zone; distorted, compressed follicles and corpora lutea; thick fibrous tissue, few connective cells; intercellular fibrillar substance massive, in many places undergone hyaline change, in others œdematous infiltration with swelling of cell bodies giving tissue myxomatous appearance; areas of round cell infiltration, lymph spaces and channels around these areas widened; cells of endothelial lining in state of proliferation; portions of tumor broken down showing foci of detritus with agglomeration of polynuclear leucocytes; some cyst like cavities filled with leucocytes; no bacteria. In a number of sections groups of medium sized round cells with large nuclei, frequently arranged around blood-vessels, giving appearance of foci of sarcomatous degeneration; but nature of nuclei and absence of mitoses leave it doubtful whether structures are sarcomatous. Interior poorly supplied with blood-vessels which are more plentiful at periphery. From these data it is apparent tumor is a fibroma with series of degenerative changes throughout its mass. The form resembles shape of ovarium, the indentation in its kidney-shaped outline most likely corresponding to the hilus. It must, therefore, be called a fibroma ovarii diffusum.

No. 46 (*Brothers*).—Patient aged forty-seven, married, abdominal swelling six years. Physical examination—hard, solid pelvic tumor median line, apparently continuous with uterus. Abdominal section several weeks later—fibroid growth of left ovary, large broad pedicle. Tumor oval in shape, weight sixteen ounces, kidney-shaped with constricted hilum, considered characteristic of ovarian fibromata, several cavities, one true cyst. Microscopical examination showed typical structure of fibroma, certain portions myxomatous or had undergone colloid degeneration.

No. 47 (*Shober*).—The author exhibited specimen of oval ovarian tumor size of large cocoanut. Growth originated from portion of right ovary, the Fallopian tube connected by part of broad ligament showed no abnormality. Tumor firm and heavy, smooth surface, capsule resembled peritoneum, densely adherent.

Section shows wavy bands of fibrous tissue, interior contains few small ill-defined cysts, no large blood-vessels. Microscopical examination shows many bands of fibrous tissue containing moderate number of nuclei, elongate and narrow, not present in uniform numbers throughout section. Some portions consist largely of long parallel fibres with few nuclei, others show considerable number of narrow rod like nuclei in elongate cells, no inflammatory infiltration. Postoperative diagnosis—ovarian myofibroma.

No. 48 (*Weir*).—Patient aged forty, nullipara, miscarriage eight years ago, slight leucorrhœal discharge, abdominal tumor with marked dysmenorrhœa. Physical examination—cervix near outlet pointing downward, uterus close to symphysis, pushed to right of median line, apparently connected with mass filling Douglas's pouch and extending into abdomen. One growth size of adult head, of stony hardness, another posterior in cul-de-sac size of orange connected with main tumor. Abdominal section shows dense adhesions; mass occupying cul-de-sac, adherent and enlarged fibroid ovary; uterus and right ovary removed. Ovarian tumor roughly spherical, smaller elongated excrescence on one side, pearly white and of stony hardness. Section shows interlacing bands of dense fibrous tissue running in all directions, one small cyst resembling Graafian follicle, no corpora lutea or corpora fibrosa. Microscopical examination shows dense fibrous tissue arranged in interlacing bands, many have hyaline appearance, fail to show any nuclei and take eosin deeply. Peritoneal cells and germinal epithelium wanting, tunica albuginea considerably thickened, small amount cortical substance forming thin stratum overlying dense fibrous tissue, several ova but no other typical ovarian elements except small cyst mentioned which fails to show any epithelium on membrana granulosa, these well defined, probably old Graafian follicle, vessels few in number. Postoperative diagnosis—ovarian fibroma, areas of hyaline degeneration.

No. 49 (*Rouffert*).—The author describes typical ovarian fibroma with twisted pedicle which was considered remarkable inasmuch as the solid tumor had developed, not at the expense of the whole ovary, but only from one of its poles, the remainder of the gland preserving its ordinary appearance.

No. 50 (*Lauwers*).—The author exhibited a fibroma attached

to outer extremity of right ovary, which in size and shape appeared normal. Tumor weighed over four and a half pounds. Microscopical examination—pure fibroma.

No. 51 (*Piering*).—The author describes a fibroma of right ovary size of two fists, on the lower median side close to pedicle was small zone of ovarian tissue, a corpus luteum and some small smooth walled cysts, with papillomatous excrescences on inner surface.

No. 52 (*Fairbairn*).—Patient aged thirty, married, three children, last confinement stillbirth. During last pregnancy suffered great pelvic pain and numbness of left leg, after delivery numbness disappeared but pain continued. Physical examination showed enlarged left ovary in rectovaginal fossa. Abdominal section revealed lower third of left ovary lodged in recess of rectovaginal fossa, weight of ovary so stretched Fallopian tube that it exceeded length of its fellow by three cm. Ovary presented covering of delicate villous tufts. Right ovary and tube normal. Tumor size of hen's egg, oval in shape, slightly nodulated. Section showed firm grayish-white surface with lighter strands of irregular meshwork; characteristic hard fibroma. Microscopical examination revealed interlacing strands of cells with oval nuclei drawn out in direction of bundles, nuclei everywhere abundant, also much wavy fibrous tissue. Typical ovarian fibroma.

No. 53 (*Ibid.*).—Patient aged thirty-nine, married, upon whom ventrofixation for prolapse had been repeatedly performed, also perineorraphy, several years previously. Physical examination demonstrated solid tumor size small cocoanut in Douglas's pouch, considerable depression of vaginal walls, growth thought to be ovarian. Abdominal section—uterus firmly adherent to anterior abdominal wall, tumor of left ovary weighing eight ounces, short thick pedicle. Growth hard, globular, ovary and small cyst on outer surface, Fallopian tube normal, fimbriated end patent. Section of tumor hard and fibrous. Microscopical examination—elongated spindle cells crossing and recrossing in all directions, fibrous stroma, plentiful supply blood channels. Ovarian fibroma.

No. 54 (*Ibid.*).—Patient aged fifty, married twenty-one years, external genitals undeveloped, no vaginal canal, menstruation never occurred. Abdominal tumor five years, markedly increased in size

last year. Physical examination—firm, smooth, round growth extending half way to umbilicus, impacted in pelvis. Abdominal section—tumor attached by broad ligament to right uterine appendage, uterus represented by cord thickness of little finger, left tube and ovary not located. Weight tumor two and a half pounds, ovoid, white, smooth surface. Ovary spread out, instead of appearing as definite mass, forming thick capsule over part of growth. Section of tumor is very hard, fibrous in appearance, fairly uniform in texture, lighter spots surrounded by fibrous bundles which stand out from cut surface. Ovarian fibroma.

No. 55 (*Ibid.*).—Patient aged thirty-four, single, painful abdominal tumor five years, dysmenorrhœa, frequent and difficult micturition. Physical examination demonstrated hard growth reaching half way to umbilicus, impacted in pelvis, slight mobility. Abdominal section—tumor right ovary, long pedicle, weight two pounds, ovoid, smooth, white shiny surface, some injected veins. Cut surface of pedicle shows mesosalpinx and edges of inner pole of ovary, Fallopian tube three inches long, fimbriated and patent lying free of growth, ovarian fimbriæ along surface toward spread-out portion of ovary. Outer covering of tumor pearly white with fibres running in various directions, from ovarian fimbriæ extending downward and inward slight depression forming V-shaped area, fibres radiating over capsule giving appearance similar to external surface of heart. Section of growth is firm, white, no evidence of degenerative change, white capsule, from appearance and continunity seeming to be from tunica albuginæ, near ovary is separable from growth but at opposite point firmly attached to underlying tissue. Microscopical examination shows tumor composed of intersecting bundles elongated spindle cells running in all directions, nuclei cut across in many different planes. Nuclei abundant, where least crowded together well marked stroma of wavy fibrous tissue. True ovarian fibroma.

No. 56 (*Ibid.*).—Patient aged twenty-seven, single, hard lump in abdomen, pain over sacral region three months. Physical examination—large, solid mass extending from pelvis to above umbilicus. Per vaginam tumor could not be differentiated from uterus and appeared to move therewith, rounded lower end in Douglas's pouch and right posterior quadrant of pelvis. Abdominal section

demonstrated tumor of right ovary weighing over four pounds, roughly globular. Attached thereto Fallopian tube three and half inches long, lying free from tumor and apparently normal, fimbriated end patent, ovarian fimbriæ traced to portion of ovary lying on surface of growth. Number of small subserous cysts about fimbriated extremity. Section of ovary showed follicles with remains of membrana granulosa, scars, dense spindle-celled stroma, much more cellular than tumor. Microscopical examination showed growth composed of irregular spindle cells, much fibrous stroma, in some portions abundance of nuclei and little ground substance, in others nuclei sparse and fibrous stroma abundant. Typical ovarian fibroma.

No. 57 (*Doran*).—The author exhibited specimen of ovarian tumor weighing three pounds, removed from patient aged fifty-two. Microscopical examination showed dense stroma of pure fibrous tissue, with distinct areas of young connective tissue. Recorded as pure fibroma of the ovary.

No. 58 (*Boyd*).—Patient aged sixty-one, single, menopause at forty, abdominal tumor two years, rapid development last three months, bladder irritable. Physical examination showed solid tumor in Douglas's pouch, evidently located in broad ligament, bulging forward on left side. Cervix moved with growth, fundus uteri could not be defined. Abdominal section demonstrated solid tumor of left ovary weighing six pounds, no adhesions, short pedicle, right ovary atrophied. Ovarian fibroma.

No. 59 (*Bissell*).—The author on opening abdomen of a woman discovered an acute anteflexed uterus and cyst size of egg in left ovary, and solid tumor of right ovary which was slightly adherent on its posterior border to sacral region of pelvis. Examination proved solid growth to be pure ovarian fibroma.

No. 60 (*Emmet*).—The author exhibited (New York Obstetrical Society) two solid ovarian tumors, one size of fetal head, other large as lemon, removed from a single woman of forty-four whose menses had ceased seven years previously. Abdominal tumor noted two years, continuous pain induced patient to seek surgical relief. The larger growth was densely adherent, which was untrue of the smaller. Examination showed both tumors to be typical fibromata which had undergone marked calcarous degeneration.

No. 61 (*Graham*).—Patient aged thirty-eight, single, poor health last two years, pelvic tumor one year, gradual enlargement. Physical examination showed large, firm, slightly movable tumor in lower abdomen, uterus and cervix normal, ascites. Abdominal section—large amount straw-colored fluid, globular growth size adult head, extensive adhesions to sigmoid, colon, pelvic brim and iliac fossa. Tumor attached to right broad ligament and Fallopian tube, no pedicle. Left ovary atrophied and hard, uterus normal, movable, no connection with growth. Death five days after operation. Post-mortem—acute fibrinous peritonitis, necrotic opening in sigmoid. Section showed tumor made of irregularly arranged anastomosing outbranching system of glistening fibrous bands, in some portions surrounding yellowish gelatinous areas. Microscopical examination demonstrated true fibroma, no trace ovarian tissue.

No. 62 (*Royster*).—Patient aged fifty-five, married, severe menorrhagia and metorrhagia many years previously, at twenty-five small growths (probably polypi) removed from cervix, menopause at fifty. Physical examination showed stony hard, smooth, movable tumor size of orange in posterior cul-de-sac, uterus normal size, pushed upward. Abdominal section—four-inch median incision, small quantity ascitic fluid, tumor dense, somewhat flattened, attached by pedicle to right broad ligament, no adhesions, left ovary atrophic. Growth weighed 451 grammes, tissue characteristic of fibroma durum, dense bands fibrous tissue, few cells and blood-vessels.

No. 63 (*Goffe*).—Patient aged thirty-five, married, pain in right abdomen extending down right leg one year. Physical examination—solid tumor right side. Abdominal section—hard, solid, symmetrical growth right ovary four and three-fourths inches in diameter, no other ovarian tissue present that side. Pedicle meso-ovary, Fallopian tube and mesosalpinx, twisted one and half times. Tumor engorged, black in places, superficial necrosis, adhesions to uterus, intestines and omentum, left ovary normal. Pure fibroma, hemorrhagic and degenerative throughout.

No. 64 (*Taylor*).—The author exhibited (London Obstetrical Society) an example of pure fibroma which sprang from outer pole of left ovary. The specimen was removed by another surgeon, from a sterile married woman aged fifty years. The case was re-

corded as one of the rarest examples of ovarian fibroma, springing from the tunica albuginea, and not from the ovarian stroma.

No. 65 (*Brothers*).—Patient aged thirty-nine, married, pain in right iliac fossa. Uterus curetted and packed with gauze, posterior vaginal fornix then incised, peritoneal cavity and pelvic organs examined. Right ovary buried in adhesions as result of pelvic peritonitis, contained hæmatoma which was incised to permit escape of blood, cavity then packed with gauze and allowed to drain per vaginam. Six years later, irregular menstruation, severe intra-abdominal pain. Physical examination showed large, round, firm tumor apparently attached to uterus filling lower abdominal median line. Abdominal section revealed fluctuating round tumor size of fetal head in right ovarian region. Microscopical examination showed hard portions of tumor to be pure fibroma with cystic degeneration.

No. 66 (*Jordan*).—Patient aged fifty-nine, mother of sixteen children, menopause nine years ago. Abdominal tumor two and a half years, gradual enlargement, distress in breathing, alcoholism, hypostatic pulmonary congestion, large umbilical hernia, ascites. Abdominal section one month later—evacuation of ascitic fluid, radical cure of umbilical hernia, large solid tumor right ovary. Section shows tissue of stony hardness, no signs of degeneration, portion of capsule of ovarian tissue. Microscopical examination confirmed section—typical ovarian fibroma.

No. 67 (*Dauber*).—Patient aged seventy-seven, large, immovable pelvic tumor causing extrusion of uterus. Abdominal section shows solid tumor right ovary. Examination reveals pure ovarian fibroma.

No. 68 (*McMurtry*).—Patient aged thirty-seven, married, abdominal tumor of recent origin and gradual development. Physical examination shows hard, nodular, movable growth in right pelvis independent of uterus, ascites. Abdominal section shows solid irregular tumor of right ovary size of orange, long pedicle no adhesions, left ovary and tube normal. Microscopical examination shows dense fibrous tissue, interlacing bands running in all directions, few nuclei, blood-vessels scanty. Postoperative diagnosis—ovarian fibroma.

No. 69 (*Vance*).—Patient aged fifty-five married twenty-five

years, never pregnant, persistent almost continuous uterine hemorrhage forty-five years, abdominal tumor fifteen years. The growth was hard, ovoid, movable, painless at first, latterly abdomen markedly increased in size, pelvic pain became annoying factor for which (in connection with uterine hemorrhage) she was induced to seek surgical relief. Physical examination demonstrated movable abdominal tumor, also hard, immovable growth completely filling pelvis. Abdominal section—ovarian tumor size of cocoanut weighing five pounds, also large uterine fibromyoma, extensive organized adhesions, operation difficult and tedious. Microscopical examination shows ovarian growth typical fibroma, some portions had undergone cystic degeneration.

No. 70 (*Ibid*).—Patient aged thirty-two, unmarried, when fourteen consulted surgeon of Cincinnati (Ohio) who removed small uterine growth (probably polyp). She had not menstruated since sixteen, in fact menses had appeared but five times in her life. Small in stature but fleshy and weighed one hundred and sixty pounds. Had been in bed fourteen weeks on account of severe pain from pelvic tumor. Physical examination shows hard growth size of orange slightly to right of median line. Abdominal section demonstrates large fibroid tumor involving uterus, both Fallopian tubes, and both ovaries. Small fibroid impinged upon bladder causing frequent and painful micturition. Microscopical examination shows fibroma of both ovaries and both tubes.

No. 71 (*Ibid*).—Patient aged fifty-seven, married, painful abdominal tumor six years, rapid increase in size last two years, ascites, intestinal disturbance from pressure. Physical examination reveals firm, nodular tumor in right side of abdomen. Abdominal section shows solid, irregular growth larger than adult head in right ovary (left ovary studded with small fibrous tumors), long pedicle, no adhesions, uterus uninvolved, large quantity ascitic fluid. Section shows cystic degeneration in centre of large growth. Microscopical examination reveals interlacing bands of fibrous tissue, typical fibroma of both ovaries.*

In the larger percentage of the cases presented (purporting to to be examples of true ovarian fibromata) in which the aid of sur-

* Some of the cases reported from Louisville (Kentucky), so far as the writer is aware, have not heretofore appeared in print [M.].

gery was invoked, the ante-operative diagnosis was pedunculated uterine fibromyomata; but, as intimated, accuracy in diagnosis in this connection is a matter of insignificant import, since the treatment is in no wise influenced thereby.

Of course it is clearly unnecessary to state that the appropriate treatment of ovarian fibroma, like that applicable to all other neoplasms involving the ovary, is invariably surgical; likewise that the operative mortality from surgical intervention should be only slightly if any greater than that which obtains after oöphorectomy for simple ovarian cystomata, which, in competent hands, is practically nil.

In the majority of the cases cited, where the patients were subjected to abdominal section before the development of complications which further endangered life, prompt and complete recovery resulted; even in occasional instances where surgical intervention was postponed until many years after the neoplasm was first detected, the ultimate outcome was recovery.

Analysis of the records demonstrates that, more often than otherwise, in ovarian fibroma the uterus and Fallopian tubes did not participate in the fibromatous process; occasionally, however, the uterus and both adnexæ were similarly affected. It also appears that, contrary to the statement of Taylor (*loc. cit.*), torsion of the pedicle occurs but rarely; likewise that the claim of Senn (*loc. cit.*) that ascites is almost an invariable concomitant symptom. is not entirely borne out by the facts.

BIBLIOGRAPHY

Andrews: Northwest Lancet, St. Paul, 1892, xii, 220–222.
Andrews: Tr. Minn. Med. Soc., St. Paul, 1892, 56–61.
Bagot: Tr. Roy. Acad. Med., Ireland, 1890, viii, 322.
Baldwin: Brooklyn Med. Jour., 1896, x, 254.
Barber: Northwest Med. Jour., Minneapolis, 1892, xx, 161.
Baron: Arch. gen. de mèd., Paris, 1842, 3 s, xiv, 82–86.
Begouin: Assoc. france de chir., proc. verb., Paris, 1904, xvii, 700.
Bender and Heitz: Bull. et mem. soc. anat., Paris, 1903, lxxviii, 241–244.
Berreman: Bull. soc. belge de gynec. d. obst., Brux, 1896, viii, 169.
Bianchi: Bull. d. sc. mèd. de Bologna, 1891, 7 s, ii, 41–48.
Blin: Soc. biol., Paris, 1852, iv, 115.
Bongrand and Bernex: Jour. med., Bordeaux, 1904, xxxiv, 448.
Boyd: Tr. Obst. Soc., London (1902), 1903, xliv, 176.
Briggs: Brit. Med. Jour., London, 1897, i, 1085–1087.

Briggs: Nashville Jour. Med. and Surg., 1898, lxxxiv, 124–126.
Brothers: Am. Jour. Obst., N. Y., 1900, xli, 194–200.
Brothers: Am. Jour. Obst., N. Y., 1904, 50, 104.
Chamayon: Independ. mèd., Paris, 1898, iv, 73–76.
Coe: Am. Jour. Obst., N. Y., 1882, xv, 561, 858.
Cordaro: Rassegna d'ostel eginec, Napoli, 1903, xii, 1–10.
Crawford: Tr. Obst. Soc., London (1894), 1895, xxxv, 190.
Crisp: Trans. Path. Soc., London, 1877, xxviii, 195.
Cullingsworth: Tr. Obst. Soc., London, 1880, xxi, 276–288.
Cullingsworth: Tr. Obst. Soc., London (1897), 1898, xxxix, 279.
Darling: Brit. Med. Jour., Lond., 1904, ii, 1068.
Dauber: Brit. Gyn. Jour., London, 1907, xxiii, 9.
Deelgrange: Arch. de tocol. et de gynec., Paris, 1893, xx, 896–899.
Deelgrange: J. d. sc. mèd. de Lille, 1893, i, 321–324.
Dimitu Tchakmakoff: Contribution a l'etude des fibreuses de l'ovarie, Lyon, 1903, 107.
Doran: Brit. Med. Jour., London, 1887, i, 619.
Doran: Tr. Obst. Soc., London (1896), 1897, xxxviii, 187–212.
Doran: Tr. Obst. Soc., London (1897), xxxix, 37–40.
Doran: Tr. Obst. Soc., London (1902), 1903, xliv, 172–173.
Dubar: Bull. mèd. du nord, Lille, 1893, xxxii, 7–13.
Erck: Am. Med., Phila., 1903, v, 415.
Fairbairn: Jour. Obst. and Gynec. Brit. Emp., London, 1902, ii, 128–146.
Feis: Centralbl. f. Gynakol., Leipzig, 1894, xviii, 133–135.
Goffe: Am. Jour. Obst., N. Y., 1904, 105.
Goodhart: Tr. Path. Soc., London, 1873–4, xxv, 199.
Grafe: Centralbl. f. Gynakol., Leipzig, 1895, xix, 10–13.
Graham: Ann. Surg., Phila., 1903, xxxvii, 468–470.
Graves: Bull. Free Hosp. for Women, Boston, 1903, i, 206–210.
Guelliot: Presse mèd., Paris, 1906, xiv, 1, 30.
Horrocks: Tr. Obst. Soc., London (1894), 1895, xxxv, 192.
Ingham: Am. Jour. Obst., N. Y., 1873–4, vi, 106.
Jaboulay: Bull. soc. de. chir., Lyon, 1897–8, 24.
Jacobs: Bull soc. belge de gynec. et d. obst., Brux, 1900, x, 249.
Jacobs: Bull. soc. belge gynec. et obst., Brux, 1900, xi, 53.
Jones: Brit. Gyn. Jour., London, 1905–6, xxi, 1.
Jones: Med. Press and Circ., London, 1905, ns, lxxix, 187.
Keblukoff: Khir, Moskow, 1903, xiii, 684–686.
Labroche and Charrier: Jour. de mèd., Bordeaux, 1905, xxxv, 396.
Laidley: Am. Jour. Obst., N. Y., 1900, xlii, 661–666.
Lamvers: Bull. soc. belge gynec. et obst., Brux, 1900, xi, 131.
Lanphear: Med. Herald, St. Joseph, 1906, ns, xxv, 202.
Ledoux: Jour. de sc. mèd. de Lille, 1900, ii, 327–331.
Ludlam: Clinique, Chicago, 1894, xv, 326.
Ludlam: *Ibid.*, 432.
McDowell: Phila. Med. Jour., 1898, ii, 1319.
McGuire: Buffalo Med. Jour., 1904–5, ns, xliv, 294–297.
Maclean: Jour. Obst. and Gynec. Brit. Emp., London, 1903, iv, 479–482.
Mallett: Am. Jour. Obst., N. Y., 1906, liii, 100.

Mauclaire: Compt. rendus. soc. obst. gyn. et pediat., Paris, 1903, v, 170.
Millot-Carpentier: Union mèd., Paris, 1890, 3 s, xlix, 519–523.
Morely: Bull. soc. anat., Paris, 1898, lxxiii, 617–619.
Odintzoff: Vratch, St. Petrsb., 1901, xxii, 677.
Pantock: Brit. Gynæc. Jour., London, 1892–3, viii, 312–314.
Pascal and Pillet: Bull. soc. anat., Paris, 1898, lxxiii, 240–243.
Pestalozza: Bull. d. soc. tosc di ostel et gunec., Firenze, 1902, i, 16.
Petel: Echo mèd. du nord, Lille, 1898, ii, 306–309.
Peterson: Am. Gynæc., N. Y., 1902, i, 45–68.
Peuch: Gaz. d. hôpitaux, Paris, 1905, lxxviii, 300, 339.
Pichevin: Bull. et mem. soc. obst. et gynec., Paris, 1897, 215–219.
Pichevin: Jour. de mèd., Paris, 1897, 2 s, ix, 437.
Piering: Prag. med. Woch., 1900, xxv, 501–502.
Pinzani: Gynecologia firenze, 1905, ii, 368–376.
Pocherat: Bull et mem. soc. chir., Paris, 1902, xxviii, 202–204.
Pokrovsky: Khir, Moskow, 1900, vii, 61–70.
Polk: N. Y. Med. Jour., 1885, xli, 705.
Postemski: Riforma med., Napoli, 1894, x, Pt. III, 494–498.
Powpow: Tr. Edinb. Obst. Soc., 1881–2, vii, 85–87.
Purslow: Lancet, London, 1897, i, 1398.
Recazeno: Rev. espec. med. la oto-rino-laryng espan, Madrid, 1904, vii, 285–294.
Rindfleisch: Zentralbl. f. Gynakol., Leipzig, 1906, xxx, 209–211.
Roberts: Tr. Obst. Soc., London (1897), 1898, xxxix, 9–12.
Rocher: Rev. mens gyn., obst. et paed., Bordeaux, 1900, ii, 274–316.
Rogee: Ann. de gynec. et de obst., Paris, 1894, xlii, 382–386.
Rouffard: Bull. soc. belge gynec. et obst., Brux, 1900, xi, 34–35.
Royster: N. Y. Med. Jour., 1903, lxxvii, 4.
Royster: Tr. Southern Surg. and Gynæc. Assn., 1902, Phila., 1903, xv, 389.
Schachner: Am. Jour. Obst., N. Y., 1894, xxix, 377.
Senn: Pathology of Tumors, etc.
Snyder: Physicians and Surgeons of Detroit and Ann Arbor, 1892, xiv, 306.
Spiegelberg: Monatschrf. f. Geb. u. Frauenkr., Berlin, 1866, xxviii, 415.
Stone: Ann. Gynæc., N. Y., 1903, ii, 60.
Summers: Omaha Clinic, 1890–91, iii, 20.
Swaving: Lehigh Valley Med. Mag., 1897, viii, 268–273.
Vance: Medical Age, Detroit, Mich., 1903, xxi, 685.
Vingard: Gaz. mèd. de Nantes, 1904, xxii, 1005–1011.
Warner: Cleveland Med. Jour., 1900, v, 264.
Wells: Tr. Path. Soc., London, 1858–9, x, 199.
Wettergren Eira, Stockholm, 1891, xv, 351.
Wilks: Tr. Path. Soc., London, 1857–8, ix, 299–305.
Williams: Tr. Obst. Soc., London, 1888, xxix, 247.
Zienibicki: Bull soc. anat., Paris, 1874, xlix, 673.

Genito-Urinary Diseases

THE INDICATIONS FOR SURGERY OF THE PROSTATE*

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At the end of the third month of fetal life the walls of the primitive urethra at the bottom of the urogenital sinus become thickened by an annular growth of mesoblastic tissue which later becomes differentiated chiefly into unstriped muscle. Into this tissue penetrate outgrowths of epithelium from the lining of the urethra and by their development form the prostate gland. The manner of formation of the prostate gland does not differ greatly from that of any other acinous gland. According to Piersol, who quotes from Pallin, in this early stage the glandular tissue is divided into three groups, one ventral and two dorsal, upper and lower respectively. The ventral mass is at first relatively large, but later is reduced in size, often to complete disappearance leaving the anterior commissure devoid of glands. The development of the dorsal group of glands takes place chiefly in a lateral direction, thus forming the lateral lobes of the prostate, while the glandular tissue immediately dorsal to the urethra usually persists only as a small pyramidal group of glands inclosed within the fibro-muscular tissue uniting the lateral lobes posterior to the urethra and known as the posterior commissure.

To the eye of the imagination this group of glands may be segregated and conceived as a lobe.

To gross anatomical observation it does not occur normally as a lobe any more than does the isthmus of the thyroid and it has always been my contention, therefore, that the term third or middle lobe should have no place in the nomenclature of normal anatomy. The so-called median lobe is potential rather than actual and consists in the possibilities of pathological enlargement of the pros-

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tatic tissue of the posterior commissure. Its prominence from a pathological standpoint comes from the fact that these glands lying directly beneath the bladder wall find much less impediment to growth than the remainder of the glands imbedded in the tissues and hemmed in on all sides by unyielding fascias. This freedom of growth accounts for the large size attained by this portion of the gland in many cases, and thus also for its lobe-like appearance. It is simply that a tendency for diffuse enlargement restricted in other portions of the gland by surrounding tissues finds here in the cavity of the bladder a local outlet. In my opinion, therefore, we would be speaking more accurately if we talked of median or commissural enlargements rather than lobes, and as our thinking is so influenced by our form of expression, I doubt not that some gain would accrue to current conceptions of prostatic pathology.

The pathology of the prostate is not especially remarkable for its variety. Its abnormalities arise from hypertrophies, inflammations, and less frequently neoplastic growths.

Clinically the gland derives its chief importance from its relation to the genital tract, and especially to the urinary outlet. The severity of the genital and urinary disturbances excited by disease of the prostate raises problems which are greatly complicated by the debility and functional weakness often found in these patients, partly owing to their advanced age, and partly to the complications incident to the condition.

The diagnosis of prostatic hypertrophy usually presents no difficulty and may be based usually on a history of increasing frequency and difficulty in urination, combined with the discovery by digital examination of an enlarged gland. The age of the patient and the amount of residual urine are important. Only rarely is cystoscopy necessary to establish a diagnosis. It is however, of great service in giving us information regarding the presence and degree of intravesical enlargement, the association of stone or growths within the bladder, and the condition of the bladder wall in respect to infection, sacculation or other abnormality. It is best to employ the cystoscope in all cases where no contraindications exist, such as extreme weakness or nervousness on the part of the patient, but it is more urgently demanded when it

is proposed to perform the perineal operation than when the suprapubic operation is to be done.

In a few cases acute symptoms occur without any noteworthy antecedent indications of a slowly progressing obstruction. Occasionally also, a case is encountered, which, while giving us the classical symptoms, may baffle our efforts to demonstrate the obstructing enlargement either by digital or cystoscopic examination. In such cases we must have recourse to a carefully reasoned diagnosis by exclusion. The chief conditions to be considered are stricture, chronic prostatitis, malignant disease and spinal lesions.

Strictures are readily demonstrated by instrumentation.

In chronic prostatitis our chief reliances are the history, smallness and tenderness of the gland on palpation and the absence of any noteworthy amount of residual urine. It may be well to remember that inflammation and hypertrophy often coexist and also that in rare cases a definite obstruction may arise from an inflammatory bar at the neck of the bladder, which will give all the symptoms of simple hypertrophy.

In carcinoma or sarcoma early differentiation is often impossible but symptoms will quickly raise the question of intervention. Quickness of decision is requisite.

The close simulation of prostatic obstruction by bladder symptoms arising from a lesion of the spinal cord demands that every case should be examined with regard to his station, reflexes, ocular signs and the character of his pains.

When should we operate for the relief of benign hypertrophy? The first consideration is the condition of the patient himself. We should not demand that he be a better surgical risk than is usual for one of his years. Age, with its inevitable arteriosclerosis and other senile degenerations is not in itself a contraindication. It is true that the mortality mounts somewhat with the age, but so also does the danger of the primary condition and the remarkable ability of old and feeble men to endure an operation on the prostate remains a perpetual source of surprise even to those who are familiar with the fact. The tables of Tenney and Chase, comprising 816 cases compiled in 1906, bear out this assertion and I feel that the mortality of more advanced age will be still

further reduced if we can overcome the reluctance of the physician to subject elderly men to operation unless the gravest conditions absolutely compel. The mortality of operation *per se* in the hands of a skilful surgeon is small. Fatalities are practically limited to those who are already living up to the limit of their functional capabilities and are unable to endure any additional burden.

The functional capability which chiefly concerns us is that of the kidneys. The largest toll of death after operation is exacted by uræmia. How careful we should be, therefore, to determine the renal condition of our patient. I am accustomed to emphasize this fact by saying that the postoperative treatment should begin before the operation, by which I mean that the kidneys must be made to functionate satisfactorily. If this cannot be accomplished, the question of operation should be judiciously considered, if, indeed, any operation should be done at all. In the estimation of this important feature I place my chief reliance on the daily output of urine and the specific gravity and to a much less extent in the microscopic findings and the various more or less complicated methods advanced for the determination of renal sufficiency.

Of general contraindications to operation I shall not speak as they are well recognized and always valid, but granting their absence, if we have a patient whose kidneys can perform their work with a margin of safety we have a patient who can withstand a prostatic operation.

To define definitely the degree of local trouble which renders operation advisable is not an easy task. It is well known that a certain degree of enlargement of the prostate is common in men past middle life, and from the slight enlargement which occasions no symptoms we have all grades up to that which finally causes complete obstruction. Not every patient who has difficulty in urination as a result of prostatic enlargement should be subjected at once to operation. The dangers of harboring a slowly growing prostate are not so great as to demand preventive surgery, and with care such patients may fill out their allotted days in considerable comfort. At the same time I feel strongly that many cases are sacrificed unnecessarily to too active and long continued palliative treatment.

When a man who has been experiencing difficulty in urination

begins to carry residual urine, even though small in amount, I believe the time for operation has arrived. When it becomes necessary for such a man to keep more or less urine in his bladder to enable him to void, he has definitely become a 'prostatic.' No one can tell when acute complete obstruction may supervene. At any rate, the hypertrophy and fibrosis of the bladder wall continues with increasing damage to the expulsive power. Sacculations and diverticula often form. With increasing obstruction, dilatation of the bladder and possibly incurable atony may ensue. The disastrous effects of the backward pressure upon the kidneys injures their waning secretory power and a general serious phase is added to the local condition. The entire urinary tract becomes an inviting field for infection and sooner or later this is almost sure to occur, since it is a practical impossibility to continue catheterization under aseptic conditions over any length of time. To a miserable life too frequently there comes a miserable end.

Not every patient will realize this gloomy forecast, but it is unquestionably true that it represents the outlook in the average case. Hippocrates considered prognosis the God-like art, and it is true that until we as physicians can treat our patients upon intelligent prognoses we will not give them the best that our art can offer.

Our operative resources are practically confined to two methods, both of which have as their object the removal of the prostate.

Partial prostatectomy and tunneling of the prostate have been abandoned long ago and are of interest only in connection with the historical aspect of the development of modern methods. Castration and vasectomy have suffered a similar fate. The Bottini operation and its various modifications for burning away the obstructing portion of the prostate by means of the galvano cautery are also undergoing eclipse. About the only remaining earnest advocate of the Bottini operation is Freudenberg. Some surgeons employ it in selected cases where the dangers of shock or postoperative pulmonary complications seem greatest, but its advantages are more than compensated by its uncertainty of cure and the dangers of sepsis. In spite of its apparent simplicity, experience has shown it to be followed by as large a percentage of deaths as the more complete and therefore more satisfactory operations and

this is sufficient reason for narrowing its indications to the lowest point or discarding it altogether. The recent proposal by Bier to treat prostatic hypertrophy by means of intraglandular injections of blood I would reject as uncertain, unsurgical and likely to be followed by complications as serious as those which occur in procedures which promise greater and more lasting improvement. The use of the X-ray also is almost untried and it is difficult to see much hope of success in its application.

We have left to consider prostatectomy, which may be accomplished through the suprapubic incision, through the perineum, or by a combination of both. There seems to me no sufficient reason why the combined operation should give better results than either the suprapubic or perineal method alone, and I have therefore never employed it. A suprapubic opening gives ample drainage for the prostatic bed and an additional perineal puncture not only does no good but may do harm. To my mind this is unscientific and unnecessary surgery. In certain cases the operation in two sittings may be the safest procedure. An old, debilitated patient suffering from an extreme grade of obstruction with recent acute cystitis or exacerbation of a chronic infection may be greatly relieved by a simple suprapubic cystotomy and in a very short time be transformed from a dangerous surgical risk into a relatively safe one. After improvement has taken place the prostate may be removed, preferably, by the suprapubic route. This procedure will be found necessary but rarely, and only in neglected cases. I prefer, if possible, to complete the operation in one sitting.

For some time the chief topic of interest in prostatectomy has related to the strife between extreme advocates of the perineal or of the suprapubic route of removal to the exclusion of the other. To-day I am of the opinion that here, as in so many other questions of general or medical bearing, the truth is to be found between the two extremes. We have arrived at the stage where two splendid operations are to be reconciled and not pitted against each other. In this view I am influenced both by operative conditions as I have found them, and by a general survey of my own results and those of others. Not in the slightest would I detract from the success of men who use but one method. On the contrary, I extend my congratulations that they have been able to employ it

successfully in cases which lend themselves more readily to the other method. But that they should recommend others whose experience has been slight to follow their example is, I believe, bad teaching and sure to produce bad results.

Under normal conditions the bulk of the prostate is about equally divided between the glandular tissue and the fibromuscular framework. On the basis of this fact we may divide hypertrophied prostates, pathologically, into three groups.

When the glandular tissue predominates in the hypertrophic process, we have formed the large, soft, adenoma-like prostate. Great median enlargement is most often found in this group. In most cases these glands are readily separated from their investing sheath, being less firmly anchored by fibrous adhesions than in the second form in which the fibrous tissue is relatively increased, producing a hard, and usually smaller enlargement, often bound by the firmest fibrous tissue to the surrounding sheath. The glandular type is more often met with than the fibrous variety.

About as numerous as both these groups together is the third or mixed variety of hypertrophy which shows little preponderance of either fibrous or glandular tissue.

One of the simplest and easiest operations which the skilled surgeon can perform consists in the suprapubic removal of a large glandular prostate. The presence of median hypertrophy rather assists than hinders enucleation. The large prostates are practically intravesical tumors, as they enlarge upwards in the direction of least resistance. To begin the enucleation, one has only to break through the vesical mucous membrane and a rarefied layer of muscle and fibrous tissue. Ease of execution permits rapidity and very short anæsthesia, a factor of great importance to the aged. Shock in my experience is rare. Hemorrhage is easily controlled. Drainage is free. The operation conforms in every way to surgical principles and in cases who can endure any operation at all a speedy convalescence and satisfactory result are almost assured.

I see no magic in the perineal operation which measures with the advantage of the suprapubic approach in these cases. To make use of the more cumbersome and lengthy perineal method seems to me the surgical analogue of polypharmacy, a resort to complex means of treatment, where simple means would do.

Inasmuch as we are so loudly invited nowadays to abandon the high for the low operation even in these cases let us compare the two more in detail.

The suprapubic incision passing as it does through the prevesical fat and areolar tissue opens up an area which must be carefully guarded against infection.

This step, usually so little considered, is I believe responsible for a small part of the mortality of the operation, by reason of prevesical suppuration. A slight amount of toxic or bacterial absorption may be sufficient to turn the tide against a feeble patient. To prevent this misfortune I make a clean cut through the prevesical tissue into the bladder, avoiding anything like blunt dissection or separation into layers. By thus avoiding injury to the delicate fat and areolar tissue, as well as opening up dead spaces for accumulation of foul products, the danger of infection is minimized. At the completion of the operation I do not close the lower angle of the wound tightly but merely draw it together and drain with gauze. Since adopting this method I have had no trouble with infection.

The suprapubic incision threatens no important structures. The peritoneum is so easily avoided that it cannot be considered as in dangerous proximity.

The perineal incision, it is true, passes through tissues less susceptible to infection. It possesses, however, the disadvantage of being made through an area of skin which is difficult of sterilization and also of proximity to the rectum which if accidentally wounded may produce severe suppuration of the wound. From the enthusiastic accounts of some advocates of the operation the unsuspecting beginner might be led to believe that the danger of wounding the rectum was very slight indeed. Nevertheless in the series of 530 cases collected by Watson there were 2.7 per cent. of persistent urethrovesical fistulæ, and certainly the rectum must have been wounded even more frequently. Tight packing occasionally causes ulceration into the rectum.

This unfortunate sequel is almost unknown in suprapubic cases.

Another danger of the perineal approach is the possibility of damaging the compressor urethræ, thus causing incontinence. The membranous urethra with this important sphincter is exposed in

the field of operation. Correspondingly, Watson's figures give us 3.5 per cent. of incontinence after the perineal operation. After the suprapubic operation in which the manipulations are all conducted above the sphincter, incontinence is very rare. I have never seen it. If our patient, after the operation, is unable to retain his urine, surely, his last state is worse than his first.

The high incision permits direct inspection of the bladder wall and ready operative treatment of any abnormality. Stone is present as a complication in not a few cases and may be easily removed regardless of its size. A stone could much more readily escape detection in a perineal operation, or if large could give great trouble in extraction. For this reason cystoscopy is very important before a perineal operation but makes less difference if the bladder be opened from above.

In so far as the object of operative intervention in these cases is concerned the suprapubic operation leaves nothing to be desired. The gland is removed cleanly, either with or without the urethra. I am able to save the prostatic urethra in a few cases, and try to do so when I can. The chance of stricture is less when the prostatic urethra is not taken away. In my experience stricture has occurred in but few cases and though it required passage of sounds the functional result was good. I see no objection to the passage of sounds as a preventative of stricture and always bougie my patients at about the end of the first week or ten days and have had no bad results from it so far as I could observe. The occasional passage of a bougie thereafter is a safe and not unwise procedure.

The practice of leaving the ejaculatory ducts, as may be done in the perineal operation, is unnecessary in the great majority of cases. Not only are many patients beyond the age when the ducts are of any functional value but it is questionable whether in younger and sexually potent men, the preservation of these ducts has anything to do with the preservation of the power of copulation. Vasectomy at other points does not destroy this power and it is certainly not proved that removal of the ducts, where they course through the prostate, may have this undesirable effect.

To my mind the diminution or loss of sexual power which often occurs is due rather to the loss of that important sexual organ,

the prostate itself. Unless it be specially desired to preserve the power of procreation, therefore, I do not think it necessary to save the ducts and certainly it is no argument to use the perineal route generally in cases more suited to the high operation.

It is undeniable that in the vast majority of cases, the suprapubic method takes much less time than the perineal, necessitating shorter anæsthesia and smaller amounts of anæsthetic. This can have only a favorable influence on two of the most important causes of death, namely, uræmia and pulmonary complications.

On the other hand these large, soft prostates furnish the cases that are most difficult from the low approach, consume the most time, require the most anæsthetic, effect the most tissue destruction, and it is only fair to assume, furnish the majority of complications of perineal statistics. In view of the evident superiority of the suprapubic method in these cases, I am not a little surprised that anyone should be deterred from following the dictates of reason by any possible statistics, misleading as they often are.

The technic of suprapubic prostatectomy which I employ has been described elsewhere and needs no repetition at this time.

In the group of small fibrous, sclerotic hypertrophies I feel just as strongly that the perineal operation is preferable. As Moore expresses it, we must "fit the operation to the case instead of fitting the case to the operation." Here the gland constitutes a subvesical, rather than an intravesical tumor. The overgrowth of fibrous tissue often forms intimate and firm adhesions to the prostatic sheath, making a finger enucleation difficult and destructive. Here the careful perineal dissection under the guidance of the eye is more successful. I do not favor a blind perineal enucleation, but believe that cases which may be successfully treated in this way are more suitable for the suprapubic method.

In the cases of mixed hypertrophy, which, according to the figures of Albarran and Hallè, constitute about one-half the entire number, I would be guided by the size, consistency and presence of median enlargement, using the perineal route for the smaller prostates lying low in the perineum which are relatively firm and fixed to the examining finger; while for those softer, higher glands, especially when complicated by median hypertrophy I would employ the suprapubic incision.

When it is especially desired to spare the procreative function, I would resort to the perineal method, preserving the duct bearing portion of the posterior commissure in accordance with the method devised by Young.

Inflammatory disease of the prostate but seldom requires operative intervention. Occasionally abscesses form both in the simple and in tuberculous infections and these collections of pus, if they do not rupture spontaneously into the urethra, require incision and drainage.

In addition to these cases there is a small group, usually due to neglected treatment, sometimes to the refractory nature of the process, in which inflammatory thickening and fibrosis about the bladder outlet gives rise to an obstruction to the passage of urine. In rare cases the obstacle to urination may be as great as is found more commonly in the benign or malignant enlargements. This condition occurs more often at an early age as compared with hypertrophy or tumor and the patient is better able to endure surgical treatment than those who come to us in later years with degenerated organs and lowered vitality.

However, I am inclined to deprecate the advocacy of prostatectomy for chronic prostatitis. I fear, if the tendency be unchallenged, that much useless or worse than useless surgery will be done in these cases. The possibility of saving the ducts is seductive, but I have already pointed out that this is no guarantee of the preservation of sexual function. The patient should always be apprised of this danger and should not be encouraged to resort to operation unless conservative treatment is exhausted without overcoming the obstruction to urination. If it becomes necessary to operate, the perineal method is preferable.

Of the different perineal operations I prefer the careful open dissection devised by Proust of the French school and later improved by Young. I have found Young's technic and instruments very satisfactory.

Malignant disease of the prostate is not uncommon and constitutes from ten to twenty per cent. of all cases of enlargement which come to clinical observation. The prognosis of sarcoma is hopeless and needs no special operative consideration, though occasionally palliative operations will prolong life. Fortunately it

is rare and I can recall but one case in my own experience. Carcinoma is far more frequent and arises usually in a previously enlarged prostate. It is slow in spreading owing to the tough fibrous investment of the gland and herein lies our chief hope of curing the disease. The prognosis is good when the disease is discovered only microscopically after removal. When the growth has gone beyond the confines of the gland I believe operative intervention is of little avail, though the efforts of Young in this direction are praiseworthy and may accomplish some good in a few cases. When there is marked obstruction to the passage of urine, I would make a suprapubic fistula for the relief of this symptom. I would not feel justified in subjecting to more radical operation cases of prostatic carcinoma in which the growth was demonstrable beyond the limits of the gland.

Before closing I must allude to the chief dangers associated with prostatectomy and their prevention.

It is noteworthy that in general ever since prostatectomy was devised the latest statistics have been the best. I attribute this partly to the operative improvements and increased skill and experience of the operators, but also in no small degree to the more careful selection of patients and to the care bestowed upon the details of treatment other than operative.

The intelligent appreciation of the value of water has saved many lives. Water before the operation improves renal function and flushes out waste and toxic products. After operation it acts beneficially on both the circulation and the kidneys. As uræmia is the chief danger and responsible for about one third of the entire mortality the importance of this agent can hardly be overestimated. Water is the best diuretic. It is advisable to begin continuous enteroclysis immediately after the operation, and as soon as the stomach becomes retentive the patient should be encouraged to drink copiously of water. The first sign of diminution in the quantity of urine after operation should be the signal for hypodermoclysis, or in more desperate cases, intravenous infusion.

Shock is best prevented by light anæsthesia and quick, skilful operation, but when present may often be overcome by intravenous infusion of salt solution. I see so little shock after prostatectomy that, personally, I am inclined to minimize its importance.

Too great stress cannot be laid upon the importance of careful nursing. Experience, intelligence, kindness and tact are necessary.

These essential qualities are very rarely found in a male nurse, but far more often in the female nurse. I therefore, always insist that my patients shall have a female nurse. The woman who is too fastidious to learn to nurse these cases is not properly constituted to make a good nurse for any case. It is her duty not only to keep careful watch over the patient's general condition, to attend to routine after-treatment and to be on the lookout for hemorrhage or other accidents, but also by the use of cold or hot applications, gentle massage, the skilful use of pillow supports, the constant radiation of a diplomatic, sympathetic optimism, in short by all the *finesse* of nursing to aid these patients to pass through the trying hours following operation. Such care in a series of cases will mean some life saved.

Postoperative pulmonary complications are rendered less common by light and quick anæsthesia and care against sepsis, against which we must invoke general surgical principles.

Hemorrhage should not occur with proper technic. Packing is the safeguard and care should be taken to prevent straining to move the bowels by appropriate measures against constipation.

Sitting the patient up and getting him out of bed at the earliest possible moment are of the greatest stimulating value to the circulation and to the kidneys. It also avoids the pronounced tendency to hypostatic congestion and basal bronchopneumonia, common in these elderly patients.

Epididymitis is an occasional complication, but seldom serious. It is claimed that passing sounds is likely to set up this inflammation, but I have passed sounds routinely in my cases and have not seen it except in one case which was complicated by a prostatic calculus.

As to the question of the anæsthesia to be used, I do not think that this has much, if any, bearing upon the occurrence of uræmia following a percentage of these operations. Uræmia, in my judgment, is the result, chiefly, of the effect of the operation upon the nervous system and this in turn upon the vasomotors. Nitrous oxide and oxygen, spinal anæsthesia and local anæsthesia,

may have a place in this operation but with ether generally, and chloroform occasionally, I have no fault to find.

CONCLUSIONS

1. In the absence of general contraindications prostatectomy is indicated in benign hypertrophy whenever there is a constantly present residual urine.

2. Suprapubic prostatectomy is indicated in large soft, glandular enlargements and when the hypertrophy is complicated by calculus, or operable disease of the bladder wall.

3. Perineal prostatectomy is indicated in smaller fibrous hypertrophy and when preservation of procreative power is a desideratum.

4. Selection and the pre- and postoperative handling of the patient is of equal importance with the type of operation.

5. Chronic prostatitis is operable only after exhaustion of conservative measures and in presence of definite obstruction to urination.

6. The hope of cure of malignant disease of the prostate lies rather in early operation than in more radical operative measures.

Pædiatrics

OBSERVATIONS ON INFANTILE TYPHOID FROM THE BABIES' WARD OF THE SYDENHAM HOSPITAL

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Infantile typhoid is not uncommon. It is frequently overlooked. It may run a short course terminating in ten days or less, or a long course of several weeks, resembling the adult type. Continued fever in infancy, with gastro-intestinal symptoms, should cause a suspicion of typhoid. It is important to disabuse the mind of adult typhoid symptoms because the clinical manifestations of infantile typhoid are peculiar to this particular age.

The following points will aid in the diagnosis:

The Blood.—There is a marked diminution in the number of leucocytes, hence *leucopœnia* may be looked upon as a constant factor in typhoid. The white blood-cells were as low as 4000, and in several of my cases recently seen were 5000. It is *absolutely pathognomonic of typhoid* and ranks equal with the Widal reaction.

Widal.—The Widal reaction is negative early in the disease, and appears after the first week or usually at the beginning of the second week. A positive Widal reaction in a dilution of 1–20 means typhoid.

Be sure that the child has not had a previous attack of typhoid. A Widal may be present years after the disease terminated.

Special Symptoms.—Infantile typhoid has no characteristic stool. In some cases it is yellowish, lumpy, malodorous, resembling scrambled egg. Other children have mucus and jelly-like masses with shreds resembling membranes; while still other children have a greenish-black liquid stool. The stool is also lienteric at the beginning, depending mostly on the diet and showing the evidence

of intestinal indigestion. Thus the stool *per se* was not uniform in all cases and did not show characteristic appearances.

Flatulence.—There is considerable flatulence.

Stomach.—Vomiting may or may not be present. The infant refuses food, is very thirsty, and seems easily satisfied.

The Abdomen.—The abdomen is markedly distended and tympanitic on percussion. Meteorismus was present in nine out of ten cases.

Eruption.—Rose-colored spots, disappearing when pressed with the tip of the finger, were seen on the thorax and abdomen. The eruption appeared in crops lasting three to six days. Some spots could be seen after ten days; the latter instance was rare.

Tongue.—The tongue is thickly coated with a gray fur. The edge of the tongue is clean, and shows enlarged swollen papillæ. At times the tongue (all but the tip) has a very whitish fur, remaining at least ten days. Severe cases have a very dry and glossy tongue.

Teeth and Lips.—The teeth and lips are dry and require oiling. The mouth is very dry and water is greedily and eagerly taken.

The Spleen.—The spleen is not always palpable, a very difficult organ to feel, especially when the abdomen is distended, during the first ten days of the disease.

The Liver.—The liver is not always enlarged.

The Lungs.—Sonorous and sibilant râles are frequently heard. Rhonchi may be present on both sides without any evidence of consolidation, but frequent examinations must be made. If the respiration-pulse ratio of 1 to 4 or (25–respiration—100–pulse) is disturbed, then pulmonary complication should be suspected.

The Heart.—Weakness or muffled heart sounds are usually noted late in the disease, especially if severe toxæmia existed. Early in the disease no special cardiac symptoms exist. A systolic murmur was heard in every case of severe typhoid.

Urine.—The importance of urine examination must not be forgotten. When it is impossible to obtain a specimen, then a sterile catheter should be used. The Diazo reaction, if positive, strengthens the diagnosis of typhoid early in the disease.

The urine contains urates, phosphates, and in one case, still

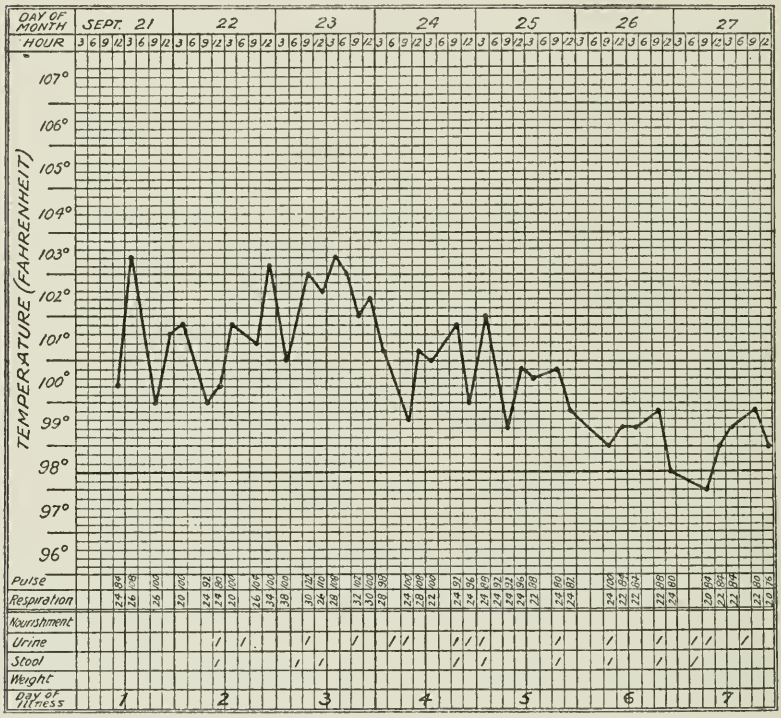
under observation in the hospital, a strong indican reaction was present.

The effect of the typho-toxine gives rise to an acute renal congestion and then we have albumin, some hyaline casts, leucocytes, and a few red blood-cells present. It is important to watch for the appearance of secondary nephritis.

Fever.—Infantile typhoid presents no regular type of fever. The following cases will illustrate this point:

Case I.—E. K., 8 months old, was admitted to my service on September 21. He had been ill about four days. The fever ran an

Fig. 1.



Case I.

irregular course for five days, and then remained normal; Widal positive; Diazo positive; leucocytes, 6000; lymphocytes, 48.5 per cent.; polynuclear neutrophiles, 51.5 per cent. As convalescence appeared and the temperature returned to normal, the leucocytes, ten days after admission, were 7400; polynuclears, 60 per cent.;

lymphocytes, 40 per cent. The case was discharged three weeks after the date of admission; leucocytes, 9200; polynuclears, 69 per cent.; lymphocytes, 31 per cent.

The stool was of a yellowish liquid character, and malodorous. The spleen was not enlarged. A systolic murmur was heard at the apex of heart.

Case II.—G. H., 18 months old, was admitted to the Babies' Ward, October 5, 1909, having been ill less than one week. A

Fig. 2.

Case II.

roseolar eruption on the thorax and abdomen led to a suspicion of typhoid. Widal positive, in a dilution of 1–20; leucopænia was present; leucocytes, 6500; polynuclears, 47 per cent.; lymphocytes, 52 per cent.; eosinophiles, 1 per cent. An irregular type of fever lasted three days, when convalescence was established. The stool was yellowish of the scrambled egg variety, and the bowels moved no more than once or twice a day. The spleen was not palpable. The case was discharged cured.

A study of the fever charts in the two cases here given is instructive. Here it can be said that we do not have the stepladder type of fever so common in adults, but that we have an irregular febrile curve. There are some cases that resemble the adult type of fever. An interesting sequelæ of the infant 18 months old (the case above cited) occurred one week after admission to my service at the hospital. An older brother about 7 years old complained of headache, fever and vomiting. A blood examination was made which gave a positive Widal reaction. There was also marked leucopænia, the white cells being 5400. Thus the diagnosis of typhoid fever in the infant was strengthened by the occurrence of typhoid in the older brother.

The clinical data and special blood work were made by Drs. Clurman and Freund of the Resident Hospital Staff, to whom I am indebted.

NOTES ON THE DIAGNOSIS AND TREATMENT OF CONGENITAL SYPHILIS

BY GODFREY ROGER PISEK, M.D.

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The diagnosis of congenital syphilis is comparatively easily made when the typical group of symptoms is present. That the text-book picture is too often sought for by the practitioner and the diagnosis discarded because the infant does not show all the characteristic phenomena, is the experience of the writer. Some of the syphilitic infants do not present more than a few of the symptoms of the disease and their true malady is in consequence unsuspected and the treatment is misdirected.

The prognosis is greatly influenced for the better if the treatment is instituted promptly and with due regard to the severity of the symptoms present.

Many of the laboratories are now equipped and stand ready to make the Wasserman-Noguchi test in suspected cases for the practitioner, so that it is not necessary to delay treatment until the blood and nutrition have been badly impoverished.

If the virulence of the infection has been such as to produce the signs of the disease at birth, the infant is underweight, emaciated, and the expression is senile. On the face and extremities is seen the maculopapular syphilide or pemphigus and the picture is heightened by a persistent coryza and excoriations at the mucocutaneous folds. A straightforward syphilitic history is not readily obtained from the average parent, but the history of miscarriages in the early months or the previous birth of a dead fœtus is excellent corroborative evidence.

It is to the infant that is born apparently without any signs of the disease that attention needs to be called. The well-developed coryza and exanthem may not appear until the second or third month, while the premonitary signs, which would lead the physician to suspect lues are already present. The following are types of

infants that have come under our observation and that proved to be syphilitic either by the blood test or the further development of symptoms.

Infants that show failing nutrition in spite of a good supply of breast milk and whose blood gives evidences of a simple anæmia shown by a deficiency of hæmoglobin and erythrocytes.

Infants with a history of stubborn hemorrhage from the umbilical stump and from the mucous membranes of the alimentary tract.

Infants that have convulsions, that are markedly anæmic, that have a splenic enlargement, and that are extremely restless and sleepless in spite of apparently good digestion as shown by the stools.

Careful inquiry in this class of cases may elicit a confirmatory history, or the therapeutic test may give results that will establish the diagnosis. If the blood test is available this should be made.

When an infant is suffering from a coryza that does not subside under the ordinary treatment, a careful examination should be made to rule out the presence of adenoids. This may be done by direct palpation of the nasopharyngeal space by the little finger of the examiner or in some cases by a direct view, using a palate hook. In rhinitis the symptoms are visible on the pharynx and palate while in syphilis no effects are shown in this region. On the other hand the syphilitic coryza affects especially the inferior turbinate causing hypertrophy of this structure and inflammation of the remainder of the nasal mucous membrane. This may be seen by examination with an infant's nasal speculum. The discharge in leutic infants is thin, serous and sometimes blood tinged. The evidence is more striking if the cry is hoarse and the snuffling is persistent in spite of a nasal cleansing. The nasal symptoms nearly always precede any involvement of the skin and therefore correct interpretation of this symptom may lead to an early diagnosis and treatment.

Important to remember is the fact that no exanthem need ever appear to confirm the diagnosis. The eruption is usually seen during the first three months of life and the disc-like, somewhat raised areas, appear principaly on the lower extremities, the flexor surfaces of the upper extremity and on the face, while the trunk is

comparatively free. This syphilide appearing rather late is usually polymorphous in character and may retain a macular appearance only, or, on the contrary, large papules may be present. The color changes are distinctly helpful, the rash turning from a red, through a dirty brown to a ham color. Pyogenic infection is responsible for the pustular forms so often seen. Again the bleb-like, pemphigoid eruption which develops on the soles of the feet and the palms of the hands seems to be puzzling when no other symptoms of syphilitic infection obtrude themselves. Their size, which is usually much larger than the eruption of varicella, their location and appearance early in life and especially their purulent or bloody contents, should stamp them with their right significance. Later their rupture and the ragged desquamation make a picture which is seldom incorrectly interpreted. Areas of skin infiltration brownish-red in color, which histologically seem to result from disease of the vessel walls, are especially significant if found in connection with a persistent coryza and failing nutrition.

A case that recently came under observation showed no other signs of lues than a specific coryza and a syphilitic paronychia. The trophic disturbance in the matrix of the nail tends to produce exfoliation or ulceration of the proximal portion while the distal end is always abnormally friable. Another helpful diagnostic sign is the seborrhœic cap which is formed in some syphilitic infants and which differs from that resulting from eczematous disease in that it is darker, harder and firmer, that it can be peeled off in areas without causing hemorrhage and that the derma beneath is dry and has the characteristic syphilitic copper color.

Radioscopy, if practised in suspected or obscure cases, will be of great importance in determining the presence of a syphilitic osteochondritis as pointed out by Wegner's studies. Even before this change has taken place periosteal involvement alone may be detected in a certain number of infants, quite early in the disease.

In suspected cases of late hereditary syphilis this procedure should not be omitted if the blood examination is impractical, as the bone lesions in the short bones of the digits are often extremely puzzling. A so-called tuberculous dactylitis may prove to be specific and amenable to treatment. The tuberculin tests will also prove of value in this connection if negative reactions are obtained.

Muscle disturbances, causing pseudoparalysis of the flaccid or spastic variety, may occur in congenital syphilis and these must be differentiated from the paralyses which occur in rhachitis and scurvy. In this connection it is also well to bear in mind that rhachitic changes may occur with the malnutrition of syphilis. In the absence of a complete history, or of well marked symptoms, a radiograph would materially assist in distinguishing these bony changes from a syphilitic osteochondritis. Further safety might be sought in the therapeutic test of raw food and fruit juices in infantile scorbutus.

Late hereditary lues, like typical cases of syphilis in the first weeks of life, are not so confusing and will not be considered here. The Hutchinson triad plus the effects of the spirillosis on the bridge of the nose or on the tibia, the fissures at the angle of the mouth, or a keratitis, will fix the diagnosis.

Treatment.—The treatment all authorities are agreed should be mercury, but the form and manner in which it is given should be carefully planned and rigidly followed: virulent infections requiring treatment that will act speedily and as rapidly as possible gain control. Therefore, in infants who are born with the exanthem, the mercury should be administered hypodermatically and for this purpose the writer has used with good results daily injections of bichloride of mercury prepared according to Hebra's method, that is, 1 per cent. mercuric bichloride in a .6 per cent. sodium chloride solution, $^1/_{100}$ grain is an appropriate dose for an infant. The injections should be intramuscular and deep seated into the nates. If the proper precautions are taken, no bad local effects are observed. After five injections it is usually possible to continue the mercury treatment as in the milder cases outlined below.

If the disease is at any time complicated by an intercurrent disease, as pneumonia, the injection method will be found to favor a restitution to the original status by improving the specific condition of the patient. When mercury is given by mouth the protiodide is an exceptionally good salt to use as it is least apt to disagree, or to cause annoying diarrhœal stools; beginning with $^1/_{12}$ grain three times a day, the dose is increased to $^1/_6$ grain until the symptoms subside.

In conjunction with the internal administration, bichloride

baths in the strength of 1 to 1000 are given daily for ten minute immersions, unless there is an unusual amount of ulceration on the skin. Inunctions are not necessary when the protiodide of mercury is given internally. The coryza, however, is ameliorated by instilling adrenalin solution 1 to 5000 into the nostrils until the mercury becomes effective. Mothers' milk must not be depended upon as a vehicle for the administration of mercury, for we have never found that the infant received any of the drug in this way. Condylomata, if extensive, are cauterized once with chromic acid in a 50 per cent. solution carefully applied to the growth only, and later silver nitrate, 1 drachm to the ounce, is used daily until the growth disappears.

The active treatment is kept up until the skin is clear and the nose is no longer obstructed. Dover's powder is not necessary as a controlling agent, as is often necessary with calomel treatment.

The specific medication should be continued thereafter for eighteen months, and may be divided into periods of six months each,—during the first six months the drug is given twice a day, omitting it entirely in the third month and last month. It is given in the same dosage every other month during the second period and once a day in the third and sixth month of the last period. Following such a definite plan we have not observed any luetic symptoms appearing in later childhood. Of paramount importance is the nutrition which is best promoted by keeping the child if possible on the breast of its mother. (A wet nurse should not be employed.) If artificial feeding is resorted to, the feedings must be carefully adapted to the physical needs of the particular infant. Iodide of potassium need not be given and indeed is detrimental to success, except in cases of late hereditary syphilis when it may be combined with the mercury as in the tertiary stage of acquired syphilis.

Parasitology

HYMENOLEPIS NANA IN THE UNITED STATES WITH REPORT OF TWO CASES

BY WILLIAM H. DEADERICK, M.D.

MARIANNA, ARKANSAS

SYNONYMS: *Tænia murina, T. ægyptiaca, T. nana, T. minima, Diplacanthus nanus, Hymenolepis murina.*

This, the smallest of the tapeworms infesting man, was found for the first time in the human being by Bilharz, in Cairo, in 1851. At a postmortem on a boy who had died of meningitis he recovered a "countless number" of them. The first American case is that of Spooner reported from Philadelphia in 1872.

Since the collection from the literature of 118 cases by Ransom [1] in September, 1904, fourteen cases have come to light. These 132 cases are distributed by countries as follows:

Italy	75	Russia	3
United States	31	Japan	2
Philippines	6	Siam	2
South America	4	England	1
Germany	4	Servia	1
Egypt	3		

The cases from the United States may be tabulated as follows:

Philadelphia, 1872, Spooner [2]	1
Galveston, Texas, 1902, Moore [3]	1
Charleston, S. C., 1902, Stiles [4]	1
Macon, Ga., 1902, Stiles [5]	3
Washington, D. C., 1903–04, Hygienic Laboratory [6]	12
Amarillo, Texas, 1903, Magnenat [7]	4
Fort Porter, N. Y., 1904, Hallock [8]	2
Statesville, N. C., 1906, Stiles and Garrison [9]	1
Marianna, Ark., 1906, Deaderick [9]	4
Brooklyn, N. Y., 1906, Lambert [10]	1
Marianna, Ark., 1907, Deaderick [11]	1

To these thirty-one cases I am able to add the two following which have recently occurred in my practice.

Case I.—E. M., white, male, age 7. His parents are living and the family history is negative. He was born in Tennessee but has been living in this section of Arkansas for several years. He has had several attacks of malaria, the last of which occurred during the summer of 1908, and for which I treated him. He had pernicious malaria in 1906 and in 1907. In the summer of 1908 his face remained swollen for about three days from an unknown cause. On August 15, 1909, his face began to swell, the right eye almost closing, then the left. I saw him August 20. His temperature, pulse, and respiration were normal. The examination of the thoracic and abdominal organs was negative. There was œdema of the feet and ankles, and, to a slight degree, over the tibiæ up to the tuberosities. The face was puffy, the upper lip and the right eyelids swollen, and the left eye almost closed. The bowels were acting two or three times daily and the appetite was good. He complained of headache and frequent spitting, but no nausea, vomiting or abdominal pains. His mother said that he talked and gritted the teeth during sleep, was nervous, easily frightened, trembling when scared, and had spasms almost every time he had fever. The blood examination showed no malarial parasites, the hæmoglobin 90 to 95 per cent., and the eosinophiles 8.2 per cent. The examination of the fæces showed numerous ova of *Hymenolepis nana*. The night of August 20, he was given a dose of salts at bedtime. The next morning early he was given twenty drops of oleoresin of male fern on an empty stomach and at 9 A.M. another dose of salts. Two actions were examined at 11 A.M. and found to contain several hundred specimens of *Hymenolepis nana*. On September 1, fæces were again examined and ova being found the treatment was ordered repeated. The symptoms abated rapidly after the first treatment.

Case II.—O. M., white, male, age 11, a brother of the preceding and has been sleeping in the same bed with him for several years. His mother said that he has had about ten attacks of pernicious malaria but no malaria whatever for two years. On

August 30, 1909, a prominent red swelling occurred on the right leg at the junction of the middle and upper thirds, this swelling itched considerably. Soon after, swellings occurred on both forearms which itched only slightly. During the night the upper lid of the left eye became decidedly œdematous. The following morning he was nauseated but did not vomit. I saw him September 1. The temperature and pulse were normal, the heart and lungs negative, the spleen and liver were not enlarged, and the bowels were regular. There was œdema of the hands, feet, and legs up to the knees, of the eyelids and upper lip. The blood examination did not reveal malarial parasites, the hæmoglobin was 90 per cent., and the eosinophiles 7.8 per cent. Microscopical examination of the fæces showed numerous ova of *Hymenolepis nana* and of *Ascaris lumbricoides,* and *Trichomonas intestinalis.* The same treatment given his brother was ordered for him, with the addition of two grains of santonin. Dr. O. L. Williamson and I examined the fæces about 11 A.M. on the day of treatment and recovered a number of *Hymenolepis nana* but no *ascarides.* The œdema disappeared rapidly.

An examination of the fæces of a brother of these two boys who slept in a bed to himself was negative.

It is probable that, in tropical and subtropical regions at least, *Hymenolepis nana* is the commonest tapeworm of man. It has been estimated that in Italy and Sicily ten per cent. of the children of the lower classes are infested with this parasite. Of about 3500 persons examined by members of the United States Hygienic Laboratory staff for intestinal parasites 16 were found infested with *Hymenolepis nana* while only 2 harbored *Tænia saginata.* According to my experience the dwarf tapeworm is at least twice as common in this locality as is the beef tapeworm.

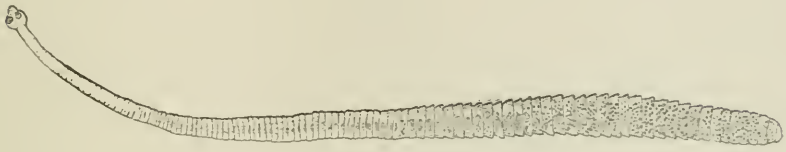
A synopsis of the United States cases, thirty-three in number, including the two herewith reported, with reference to race, sex, and age is given below:

White	18	Males	23
Colored	7	Females	5
Not recorded	8	Not recorded	5

1 to 15 years	15
16 to 30 years	3
31 to 50 years	5
Over 50 years	2
Not recorded	8

The length of my specimens of *Hymenolepis nana* has been from 8 to 18 mm.; the breadth of mature proglottides from .33

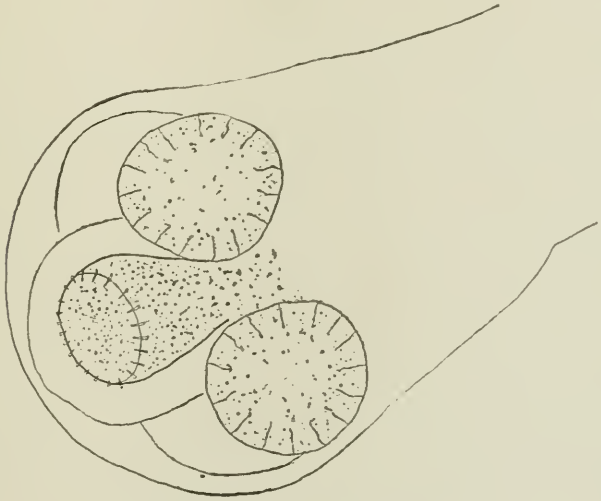
Fig. 1.



Hymenolepis nana.

to .58 mm.; and the number of segments approximately from 100 to 200. In a specimen measuring 15 mm. in length there were 185 segments. The head is subglobular, elongated, possessed of

Fig. 2.

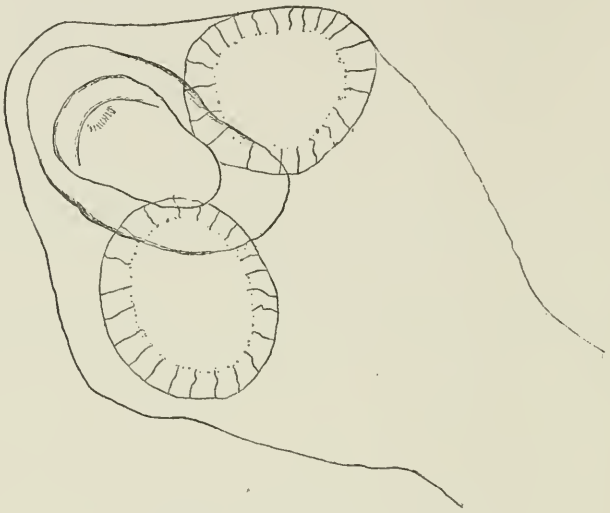


Scolex of H. nana—rostellum retracted.

a retractile rostellum surrounded by a single row of hooklets, numbering from 22 to 30, and has four suckers. The average length of the head with rostellum protruded was .34 mm.; with rostellum retracted .27 mm.; and the width of the head .27 mm.

The diameter of the suckers averaged 86 microns. The hooklets averaged 14 microns in length. The neck is slender and unsegmented. In a specimen measuring 15 mm. the distance from the tip of the rostellum to the first segment was 1.18 mm.; the

Fig. 3.



Scolex of H. nana—rostellum partially protruded.

breadth of the neck .14 mm. The segments of the strobila gradually increase in breadth toward the posterior extremity, the last 15 to 40 proglottides being usually stuffed with ova. Occasionally a sterile segment is seen between two fertile ones. The genital pores are single, near the anterior border of the segment, and usually on the left side. Each oviparous segment may contain from 80 to 180 ova. The mature ova are round or slightly oval, averaging in my cases: outer membrane, 50 x 42 microns; inner membrane, 32 x 30 microns. Between the outer membrane and the inner one which encloses the membrane is a hyaline substance which surrounds the latter like a wide halo. Within the embryo can be distinguished six hooklets lying parallel or radially. A few very delicate filaments may commonly be detected arising from opposite poles of the inner membrane and permeating the hyaline substance.

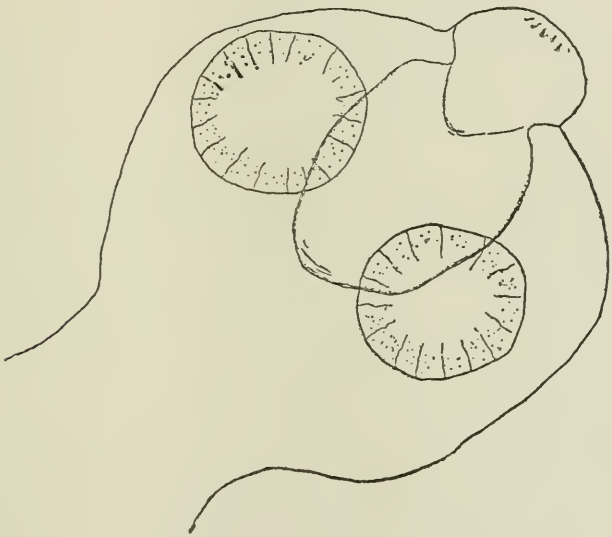
I have examined the ova in my last three cases with reference to Foster's [12] interesting observations on the movements of the embryo within the ovum but have not been able to verify them.

In eight of the North American cases there have been mixed infestations: five times with *trichuris,* once with *oxyuris,* once with *uncinaria,* and once with *Ascaris lumbricoides* and *Trichomonas intestinalis.*

Ransom [1] states that the results of a number of autopsies show that the portion of the intestine infested is a greater or less part of the ileum especially its upper two-thirds or three-fourths. The parasites may number from a score to several thousands.

In untreated cases the ova may persist in the fæces during periods varying from two months to two and a half years.

Fig. 4.



Scolex of H. nana—rostellum protruded.

The only animals besides man in which the dwarf tapeworm has with certainty been found are certain species of rats and mice.

Grassi [13] first advanced the idea that these parasites might be transmitted without the intervention of an intermediate host. He based his theory upon carefully conducted and properly controlled experiments upon rats fed with mature segments of *Hymenolepis nana.* He found that the ova did not evolve immediately into adults but entering an intestinal villus, developed there into cysticercoids, which ultimately ruptured into the lumen of the bowel

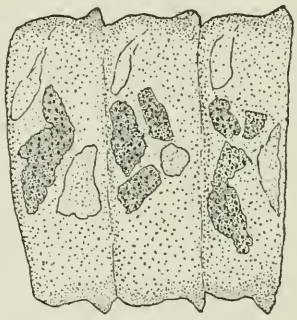
and proceeded to maturity, the intestinal wall of the definitive host thus acting in the capacity of the intermediate host. It is altogether probable that infection from man to man occurs similarly. The finding by Venuti [13] of 23 cases among 214 boys examined in an institution in Catania, while no case could be found among 100 boys of the general population, Stiles's [5] two cases in an orphan asylum, the five cases of the Hygienic Laboratory staff [6] in one ward of an insane asylum, Magnenat's [7] four cases in one family, two cases in a household previously reported by me,[9] and the two recorded above, afford grounds for this belief.

The most common symptoms in my cases have been, in the order of frequency, nausea, vomiting, œdema, headache, abdominal

Fig. 5.

Hooklets from H. nana: (*a*) lateral aspect, (*b*) view from edge.

Fig. 6.



Proglottides of H. nana.

pain, diarrhœa, dyspnœa and convulsions. Symptoms recorded by other writers are increased or diminished appetite, dyspepsia, constipation, paralysis, epilepsy, strabismus, and diplopia.

In Lambert's [10] case there was no increase in the proportion of eosinophiles although in my seven cases these cells were found in the following percentages: 11.5, 15, 9, 26, 10.5, 8.2, and 7.8.

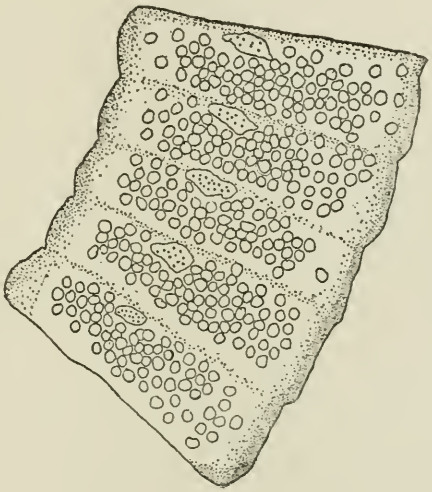
I have not found Charcot crystals in the fæces of any of my cases but in mounted specimens of the parasite have frequently noticed the formation of these crystals at points where the worm was lacerated or contused.

The diagnosis of infestation with dwarf tapeworm should be based solely upon the discovery of the parasite or its ova in the fæces. In all my cases unciniriasis was suspected and it was this suspicion that led to the examination of the fæces in my cases.

In the examination of blood if eosinophilia is detected the fæces should be examined, for this condition of the leucocytes is common in intestinal helminthiasis.

The ova are readily detected if a small bit of fæces be rubbed in a drop of distilled water on a slide, a cover-glass added, and examined with a two-thirds inch objective. The only ova with which they may be confused are those of the very rare parasite *Hymenolepis diminuta.* From these they may be distinguished

FIG. 7.



H. nana—proglottides containing ova.

by their somewhat smaller size and the absence of a granular layer between the outer and inner shells.

I have failed only once to find the parasites after the anthelmintic had been given, and then in a case in which the ova were extremely abundant—over one-hundred to each cover-glass preparation. However, in thirty cases in which the result of the search for the worm is recorded, in about one-fourth of the cases it was negative. Owing to their size, color and consistence they are difficult of detection unless properly sought for. I use a large flat "biscuit pan," 12 x 17 inches, painted black and with a row of holes punctured across one end near the bottom. The diluted fæces may be retained in thin layer, or the liquid allowed to drain

off without agitating and the parasites are easily detected against the black surface.

Prophylaxis consists of measures to prevent the access of rats and mice to the food and drink. In view of the probability of direct contagion from man to man personal cleanliness and a degree of isolation of infested individuals are advisable.

Relative to treatment I quote the following from a former article of mine: "The only remedy of value in the treatment of *Hymenolepis nana* is male fern. Manson [12] makes the statement that the worms are readily expelled by this agent, but an analysis

Fig. 8. Fig. 9.

Ova of Hymenolepis nana.

of the cases in which the results of treatment by male fern were verified by subsequent microscopical examination of the fæces shows that more than one treatment is usually necessary. There are 22 cases in which the results were thus followed up. Of these there are only five cases in which no ova were found after one treatment with male fern; in six cases two treatments were necessary; in five cases three doses were needed; in one case four or five treatments were administered; in four cases after one dose the ova were still present but no further treatment is recorded; in one case after several treatments the ova persisted in the fæces; and in two of the cases in which three doses were given the ova were yet found after the last dose. The drug should, of course, be given only after thorough purgation with salines."

REFERENCES

[1] Ransom: An account of the Tapeworms of the Genus *Hymenolepis*, Parasitic in Man, Bull. No. 18, U. S. P. H. & M. H. Service, Washington, 1904.

[2] Spooner: Am. Jour. Med. Sc., lxv, p. 136.

[3] Moore: Med. News, Feb. 6, 1904.
[4] Stiles: Illustrated Key to the Cestode Parasites of Man, Bull. No. 25, U. S. P. H. & M. H. Service, Washington, 1906.
[5] Stiles: Prevalance and Geographic Distribution of Hookworm Disease in the U. S., Bull. No. 10, U. S. P. H. & M. H. Service, Washington, 1903.
[6] Stiles and Garrison: A Statistical Study of the Prevalence of Intestinal Parasites in Man, Bull. No. 28, U. S. P. H. & M. H. Service, Washington, 1906.
[7] Stiles: Cited by, in N. Y. Med. Jour., Nov. 7, 1903.
[8] Hallock: Jour. Am. Med. Assoc., xlii, 891.
[9] Deaderick: Jour. Am. Med. Assoc., xlvii, 2087.
[10] Lambert: Paper read at Practitioners' Society, N. Y., Nov. 2, 1906.
[11] Deaderick: Jour. Trop. Med. and Hyg., Mar. 1, 1907.
[12] Manson: Tropical Diseases, New York, 1903.
[13] Ransom: Cited by (1).

FRAMBŒSIA TROPICA

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The personal experience with the disease frambœsia tropica, which has been my incentive in writing the present paper, consists in the observation of about fifty cases in Siam, in the cities of Bangkok and Phrapatoom.

Frambœsia tropica, known also as yaws, is a specific disease of slow evolution, which is propagated by inoculation, and which runs its course in three successive stages. At the site of infection (inoculation, autoinoculation, or reinoculation) a special tissue change occurs which is characteristic of the disease, and the time of appearance of the change corresponds to the first period, or that of invasion. Within a few months after the appearance of the initial lesion, a general eruption occurs, the individual members of which have the same characteristics as the primary sore. This state marks the second period, that of generalization. Still later in the disease, osteo-arthritic, periosteal, and various fibroid changes occur, together with plantar and palmar scleroderma and fissuring. This is the third, or post-eruptive stage.

Although this disease is said to have been epidemic in Scotland and Iceland in past centuries, it is now almost entirely limited to tropical and sub-tropical regions, though Castellani remarks that a disease very similar to frambœsia has recently been reported from Greece. In Africa it has been reported from numerous districts—the Soudan, the west coast of Senegambia to Angola, Algeria, the Nile Valley, Mozambique, Madagascar and Mauritius. In Oceanica, all Polynesia is infected, especially New Caladonia, Fiji, New Guinea, Loyalty, Solomon, Marshall, and Philippine Islands, and Samoa and Tonga. In Asia, reports have come from India, the coast of Malabar and Coromandel, Ceylon, Assam, Borneo, Siam, and the Malay Peninsula, Indo-China and Formosa. In America cases have been observed in the Antilles, Guiana, Costa

Rica, Venezuela, and Brazil. The above incomplete list shows the very wide distribution.

In the different parts of the world where frambœsia occurs, it is known by various but distinctive names. The term "frambœsia" (Ger. *Framboesie*) is an occidental one, and was applied to the disease because of the resemblance of the primary lesion to the raspberry (Fr. *Framboise*). *Yaws* is the English term, and *Pian* the French. In Ceylon the disease is known as *Paranghi,* in Burma as *Krocna,* in Fiji as *Coko,* in East Africa as *Buba,* and in Siam, where my own work was done, it has different names in various parts of the kingdom. In and about Bangkok it is known as *Koot'tarat;* in the Laos country of northern Siam as *Khi Kat Chin* and *Khi Mo;* and in the eastern parts and Cambodia as *Dam Bao.* There are other descriptive terms of local origin and application in great numbers.

Beside these designations that apply to the disease in general, there are other appellations that are used by various groups of natives of different districts to designate special forms of lesions or the stages of the disease. Such terms apply in some instances to painful plantar manifestations; in others to a new element appearing when the disease itself seems to have been disappearing; others to an isolated papillomatous lesion; and still others to the eruption in children. There is in the district where I worked no special designation for the secondary lesions, but some lesions that may possibly be tertiary ones are known as *payat,* an affection which corresponds even in detail to that described as a clinical entity by Jeanselme under the title *Khi Huen.*

The first description of frambœsia came from Oviedo from Santo Domingo, and the descriptive term "Frambœsia" was first used by Sauvages (1759). Charlouis, writing in 1882, used the more exact descriptive term *Polypapilloma tropicum.*

In 1848 Paulent inoculated fourteen negroes with the secretions from frambœsial lesions and all of them developed the disease, after an incubation of from twelve to twenty days. Later, in 1881, the classical experiments of Charlouis were made. Charlouis inoculated a native who was suffering with frambœsia, with syphilis, and showed that neither disease was modified by the other.

Still later Neisser, working in Java on the relationship of

frambœsia and syphilis, corroborated the work of Paulet and Charlouis, and showed, upon the basis of immunization experiments with apes and monkeys, that the diseases are distinct. A monkey that has had frambœsia is not immune to syphilis and *vice versa;* and further Castellani has shown that, by means of the Bordet-Genou or Wasserman reaction, the disease can be detected, and that the antibodies and antigens of frambœsia are different from those of syphilis. It has also been demonstrated that the infection is a general one, as is syphilis. The organism is found in the spleen and lymph-glands, and the blood obtained by splenic puncture may reproduce the disease in monkeys.

The study of the relations of yaws to syphilis has given rise to much debate as to whether yaws is an evolutionary form of syphilis, or syphilis an evolutionary form of yaws. In other words, whether Treponema pertenuis is a modified Tr. pallida or whether Tr. pallida is the result of modifications of Tr. pertenuis. This discussion is made more difficult by the fact that the two organisms are morphologically almost if not quite identical. In the orient both diseases exist side by side, and in spite of their great geographic distribution and apparent antiquity, each causes the clinical picture generally ascribed to it. In the case of syphilis, the only difference that one can see between the temperate and exotic forms is that the latter is more virulent for the white man, though it is not as a rule severe in natives, in whom, however, it may be very atypical. Whether this difference is due to an acquired racial immunity or to a racial acclimatization in the native, cannot be positively stated. It might be suggested, since in certain countries frambœsia is rather a disease of childhood, as syphilis is as a rule one of adults, that the one gives rise to a relative immunity to the other. On the other hand, the presence of congenital syphilis appears to have no power to modify the frambœsia of a child, a fact that can be explained only on the supposition that the diseases are distinct. The reactions give testimony to the correctness of the latter view.

The disease shows no seasonal prevalence, and its course is not affected by seasonal changes. It affects males and females, children and adults, indiscriminately.

It has been settled beyond a reasonable doubt that the organism which is the cause of the clinical and pathological features of

frambœsia is the *Treponema pertenuis* (Castellani). Previous to Schaudinn's discovery of Treponema *pallida,* all sorts of organisms had been described as the cause of frambœsia. Eijkman described bacilli; Pariez, micrococci; Powell, yeasts; Nicholls, and Watts, micrococci. It was not until 1905 that Castellani and Wellman, working independently, described an organism that resembled the one found in syphilis. Borne, in the following year, found the organism in nine out of eleven cases, and later, in a letter to Castellani, reported finding them forty-nine times in fifty-two cases. Castellani in 1907 reported them fifty-six times in fifty-nine cases. Since that time the statistics have multiplied at a great rate, but always point to the ultimate relation of the organism of Castellani and the disease complex. The fact that so many different organisms were held responsible for the disease in the early stages of investigation is doubtless due in part to the fact that in the ulcerated open sores of patients with frambœsia, other and various sorts of organisms are found. Cocci and bacilli are often innumerable, and at least four spirochætæ have been observed, viz., *S. refringens, S. obtusa, S. acuminata,* and *Tr. pertenuis.*

The number of treponemas in a lesion varies exceedingly. In recent lesions they are usually abundant, so that one may find one to ten or more in each field of the microscope. In older, and especially in ulcerated lesions, and those which are the seats of secondary infections, the treponemas may be difficult to find. *Treponema pertenuis* (Castellani), *seu Spirochæta pertenuis* (Castellani, June, 1905), *seu Spirochæta pallidula* (Castellani, November, 1905) is described by its discoverer as an organism from 18–20 microns in length and not above 0.5 micron in thickness. It is extremely thin as a rule, although some individuals appear thicker and more deeply stained than others. The extremities are pointed. The number of waves varies somewhat, though they are always numerous and of fairly uniform size and small dimensions. Certain variations of this picture have been described, especially by Castellani and Prowazek. Thus, certain individuals may show the typical waves in the central parts of their bodies, but none near the extremities. Others are terminated at one extremity by a knob-like or oval mass. In certain cases such ovoid bodies may be seen lying free. They measure from 5–8 x 4–6

microns, but may be larger or smaller. They contain chromatin and this may be seen collected at one point or as scattered granules. It has been suggested that these bodies represent a resting stage of the organism. Castellani states that Treponema pertenuis is very difficult to distinguish from Treponema pallida. Wellman says that it is not possible to distinguish, morphologically, the two organisms. Russel says, however, that Tr. pertenuis is slightly thicker than Tr. pallida; that the distance from crest to crest of the waves is greater; that the hollow between the crests is greater as a rule; that the waves are not so regular in height; that dividing forms are more frequently seen; and that there is a greater tendency for the organism to curl up into a loop or ball form, which is probably the resting stage. That they are separate species is assumed on the basis of inoculation and serum experiments.

Just what systematic position the treponema should occupy is not satisfactorily decided. Blanchard classified *spirochæta* and *treponema* with the *trypanosomidæ*. Novy and Knapp, on the other hand, placed them with the bacteria, upon the basis (in S. recurrentis) of transverse division; the absence of a nucleus, blepharoblast, and undulating membrane; the staining qualities of the flagella; the plasmolytic phenomena; the persistence of the spiral form under varied circumstances; and the difficulty of establishment of immunity reactions. Prowazek and Keysselitz have had evidence of longitudinal division, of an undulating membrane, and of chromidia of protozoal type; and Prowazek comes to the conclusion that one must place the organism (S. gallinarum) among the protozoa and close to the trypanosomata. Ward believes that these organisms should occupy a position in classification part way between the protozoa and the bacteria. He says, "They are certainly distinct from the spirilla, and whether they are ultimately placed among the bacteria or the protozoa, it seems clear that they will occupy a more isolated position than has previously been assigned them by the advocates of either view." Schaudinn believed them closer to the animal than to the vegetable side. They are spirally twisted flexible organisms with polar flagella, and divided by longitudinal divisions.

As in syphilis, so it is true in frambœsia, that the infection occurs at the site of some break of continuity of the cutaneous sur-

face of the body. Such breaks may appear of very minor importance. Thus in children the primary lesions are not infrequently on the elbows and knees, about the mouth, nose and the other natural body orifices. Similar extragenital primary lesions are not infrequent in exotic syphilis, but whereas syphilis is not, at least as a rule, autoinoculable, framboesia is, and from the primary lesion infection may be carried by the fingers or clothes to other parts of the body. As in syphilis, so in framboesia, the infection may be carried from person to person in a variety of ways. Clothing, bedding, drinking pipes, all play a rôle, some more frequently than others. The common oriental custom of wiping the fingers upon window ledges, door posts, chairs, and in fact any convenient article or object, is surely an important factor in the spread of this and other infectious diseases, especially syphilis and leprosy. The rôle of insects has been promulgated by many and investigated by a few writers. Castellani's experiments, while not at all conclusive, hint at the possibility of the intermediary part played by insects. Robertson, by careful attention to bodily cleanliness among yaws patients, and by smearing their sores with mercurial ointment, has prevented the spread of infection among hospital patients, and he suggests that this result has been due to the fact that flies could not become infected by feeding at the lesions. Still, the fact that in experimental infection by scientists it is necessary to scarify deeply and rub the infected material well in, seems to indicate that insect transmission is not a common method. It seems to me that the sore-picking habit is much more important. An oriental native will amuse himself for a long time picking off the dried sections of his sores, thereby soiling his hands which he wipes off on whatever object is near. With such filthy hands he fondles his children, feeds them or picks their sores, and is therefore much more able than the flies to spread infection.

As has been indicated above, the course of the disease has been divided into three stages,—those of invasion, generalization, and a post-eruptive stage; or as in syphilis, into primary, secondary and tertiary stages.

Primary Stage.—After an incubation of from two weeks to two or more months, a small papule or vesicle appears at the site of infection. At this time there is a more or less general disturbance

of health, with such symptoms as headache, gastric disorders, more or less fever and malaise, and often a very transient general papular, squamous or papulosquamous eruption. In my cases such symptoms were not regular, for while one could sometimes discover some little fever or occasionally could elicit a complaint of headache, it was more usual for a patient to deny all symptoms of ill health unless indeed very leading questions were asked. Very occasionally a patient mentioned rheumatoid pains.

The small primary vesicle may either disappear or increase in size. In the latter case, which is the more frequent, it finally becomes tipped with a yellowish point. The skin ruptures or is ruptured at this point and then the papillomatous character of the lesion becomes apparent. From this papilloma a thin, pale fluid is secreted, which in drying forms a crust. The crust becomes larger and larger by repeated additions, and assumes a rupial appearance. Beneath the rupia the lesion is bathed in its thin secretion in which are desquamated cells, a few leucocytes, and a few,—very few in my experience,—treponemata; so that in making smears to demonstrate the organism it is necessary to take scrapings from between the papillæ of the tumors. After reaching a maximum size the lesions gradually disappear under their crusts; the papillæ atrophy and the epidermis is partially regenerated, but not sufficiently to prevent scarring. The scars are at first pigmented but later become pale and often disappear.

Following this first stage, there is a more or less extended period of apparent quiescence during which the disease is generalized. This period may last a month or many months, and is terminated by a general eruption, the individual members of which are similar to those of the primary lesions; that is to say, they are plasmopapillomata.

At the time of the eruption there is again a febrile reaction with rheumatoid pains. In the lesions of this stage, as in the primary "button," the treponemata are present, and, as in the primary lesions, are chiefly in or between the papillæ of the nodules. In this stage all parts of the cutaneous surface may be involved. In my series of cases I have seen lesions on the face, feet, legs, thigh, buttocks, hands, arms, soles of the feet, palms of the hands, prepuce, angles of the mouth, about the anus, in the gluteal folds, about the finger nails, between the fingers and toes, and over the

entire back, abdomen and neck. In no case have I seen an affection of the mucous membranes or of the eyes. In every case there is a general glandular enlargement, which is most marked during the period of invasion, in the glands draining the infected area. Later, when the disease has become generalized, the glandular swelling is also general. This glandular condition is similar to that occurring in syphilis, in which, as in frambœsia, the swollen glands rarely if ever suppurate, but remain firm and moveable.

Beside these two distinct and easily recognized stages, there is a third which is indefinite, and in which the lesions are much like those of tertiary syphilis. It is the stage that is so difficult to identify, and which, more than any other feature of frambœsia, has led to its confusion with syphilis. As a matter of fact, there is still much doubt as to the unity of this tertiary stage, for the lesions appear at an age when the tertiary lesions of syphilis also appear. It is a disconcerting fact that throughout China, Cochin China, Cambodia, and Siam, these two diseases are almost equally widespread, and so the chance is present in many cases, to say the least, that either one or both are responsible for the manifestations. It is also true that if one be not extremely judicious in questioning patients, he will be able to get a history that will support a diagnosis of either disease. I have been assured by a patient that a plantar or palmar syphilide was frambœsia, as it may have been, though I still doubt it.

Jeanselme lays especial emphasis upon the painful exostoses and osteo-arthritic troubles in tertiary frambœsia. Other writers mention various sorts of ulceration of skin, palate, larynx, nares; periostitides; various forms of dermatitides and even gummata. For myself, though I have seen many such lesions, I have never felt satisfied that they must necessarily represent a stage of frambœsia. There is an additional difficulty in diagnosis in the fact that the same treatment reacts equally well in frambœsia and in syphilis. I made one experiment with a monkey, using the material obtained by curetting a sore, for inoculation. I had intended, should specific lesions develop, to inoculate again with typical syphilitic and frambœsia material. Unfortunately the experiment was negative and no more monkeys could be procured before I was transferred to another post.

Complications are rarely serious, and are chiefly confined to

ulcerations arising as a result of secondary infections. Phagedena may occasionally be met with and may cause lasting deformity or even death. One such case in my care yielded to rigid antiseptic treatment with bichloride of mercury compresses. The eosin treatment was useless in this case.

In the early stages of frambœsia there is ordinarily little trouble in arriving at a diagnosis. It is in the post-eruptive stages that the trouble comes. It may be that only animal experiments can decide some of the cases. In a general way the following outline will show the main points of difference between frambœsia and syphilis.

FRAMBŒSIA	SYPHILIS
1. Chiefly a disease of the skin.	1. Chiefly a disease of the connective tissues.
2. No falling out of the hair.	2. Hair falls out.
3. Not pandemic—tropical.	3. Pandemic.
4. Stigmata absent.	4. Stigmata classical.
5. Primary sore moderately (if at all) indurated.	5. Primary sore indurated.
6. Not hereditary in any sense.	6. Hereditary in a certain sense.
7. No visceral or ocular manifestations.	7. Visceral and ocular manifestations often present.
8. Does not affect mucous membranes.	8. Mucous membranes affected.
9. Autoinoculable.	9. Not autoinoculable.
10. Primary sore not constant.	10. Constant.
11. Reinfection possible.	11. Reinfection rare.
12. Scars permanent only after mistreatment.	12. Scars frequently permanent.
13. Two definite stages. One other indefinite.	13. Three definite stages.
14. No lesions of central nervous system.	14. Lesions of central nervous system.
15. Manifestations of one type.	15. Polymorphous manifestations.

Neisser's work has shown that (1) frambœsia is inoculable from man to the higher and lower apes; (2) that it is transferable from ape to ape, and (3) that animals that have been inoculated with syphilis are still susceptible to frambœsia. Charlouis showed that frambœsia conferred no immunity to syphilis in human beings. The serum reactions are different.

The prognosis is good. Death rarely results (I, at least, have never heard of it) in uncomplicated cases.

Prophylaxis can be insured only by personal cleanliness, by

a careful choice of servants, and by care of utensils for eating and drinking.

The most successful treatment is that known as antisyphilitic. In all of my series of cases I had excellent results with gradually increasing doses of iodide of potassium given internally, and local applications of bichloride of mercury compresses, followed by iodoform powder or ointment.

REFERENCES

Castellani: Jour. Hyg., 1907, vii, 558.
Howard: Journ. Trop. Med., July 1, 1908.
Jeanselme: Brouardel and Gilbert's Maladies exotiques, Fasc. vi, Nouveau traité de méd., 1906.
Jeanselme: Dermatologie exotique, Paris, 1904.
Mayer, Arch. f. Schiffs-u. Tropenhygiene, 1908, Beiheft I.
Plehn: Mense's Handb. d. Tropenkrankheiten, 1905, i, 60.
Robertson: Journ. Trop. Med., July 15, 1908.
Russell: Arch. Int. Med., Aug., 1908.
Ward: Studies from the Zool. Lab. Univ. Nebr., 1908, No. 90.
Wellmann: Arch f. Schiffs-u. Tropenhygiene, 1907, xi, 545.

Laryngology

DIAGNOSIS OF FOREIGN BODIES IN THE ŒSOPHAGUS AND BRONCHI

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During the last five years I have had occasion to treat 42 patients for foreign bodies of the œsophagus, and 20 for foreign bodies of the bronchi. But the total number of patients sent to me with a definite diagnosis of foreign body of one of these tubes has been much greater, at least twice that of the patients who really had a foreign body.

The diagnosis of foreign body of the bronchi or œsophagus is therefore especially difficult, and it is easy to understand that it should be so; for at the present time we have no positive means of deciding with certainty whether or not there is a foreign body in the bronchi or in the œsophagus.

If we consider the clinical signs that lead us to suspect that the patient has a foreign body in the œsophagus, we have in the first place the history of the case. That is to say, that the patient states either while eating or during his sleep that he swallowed the foreign body, or a mother tells us that her child swallowed a coin or a medal before her eyes. But the history is often far from being as clear as this; a child can very well swallow a foreign body without its parents being aware of the fact. I was lately called upon to remove a large foreign body of the œsophagus in a backward child. The accident had happened when the child was not being watched, the signs of mechanical obstruction were all that were reported, and the opinion seemed to be that the foreign body was a domino whereas in reality it was a large round piece of bone.

Nevertheless, due importance must be given to the history of the case whenever it is very clear and the foreign body has not

been eliminated in the natural way during the days that followed the accident.

The second symptom that attracts attention is the pain in swallowing, which is always very marked. It exists when food is swallowed, and the most that can generally be obtained is that the patient can take liquid food. When inflammatory signs have set in, this pain exists even spontaneously when saliva is swallowed.

But this symptom is not at all a proof of the presence of a foreign body in the œsophagus. Thus, I have in many cases searched for pieces of bone, fish bones, or nails, in the œsophagus, on account of the existence of painful swallowing, and yet there was no longer any foreign body in the œsophagus. It is the wound produced by the foreign body, the wound that was always present at the examination, that gave rise to the symptoms that led to exploration. The wounds produced by foreign bodies of the œsophagus, even sometime after their spontaneous elimination, are always very painful, and lead the physician to believe firmly in the presence of a foreign body on account of the patient's subjective sensations.

To this painful swallowing we must also add complete arrest in the passage of food down the œsophagus, of purely mechanical origin, due to the fact that a coin, for instance, is lying perpendicularly to the axis of the tube, completely obstructing its opening.

In other cases it is remarkable to observe the tolerance of the œsophagus for foreign bodies. This occurs, in particular, with round substances, or when the foreign body is parallel to the axis of the tube. There is then neither irritation nor wound, and the food passes the obstacle more or less easily by separating into different portions when it meets the foreign body.

I had occasion to remove, in one of the hospital wards, a ten centime copper coin about three centimetres in diameter that had been in the œsophagus for four years without having given rise to very much trouble in swallowing. I also removed recently from a child 2½ years old a medal that had lodged in the upper portion of the œsophagus and that did not interfere in any way with the child's eating. The father, who knew that the child had swallowed the medal, and had not been able to find it in

the stools, became uneasy, and had a radiogram made, in spite of the absence of symptoms. Great was his astonishment when the plate showed the medal lying across the upper portion of the œsophagus.

The difficulty in the clinical diagnosis of a foreign body of the œsophagus is therefore serious. Radiography is of course of great help in coming to such a diagnosis, and gives a positive result in a good third of these cases, but all foreign bodies do not stop the X-rays, as they must be of metal and have a certain size to appear clearly on the screen. Furthermore, in the lower portion of the œsophagus their shadow may very easily be confounded with that of the vertebral column.

As regards bronchial foreign bodies, the diagnosis is often more difficult still. I have had occasion to look for a great many that were entirely imaginary and where a diagnosis had been made purely on the clinical signs. That this is true is proved by the fact either that the patients are alive and all the symptoms have disappeared, or else that the diagnosis was confirmed later on.

The history of the case of a foreign body of the bronchi is also of great importance, but the most important of all is to know exactly what occurred when the accident took place. It is necessary to find out from the parents whether at the moment when the child swallowed the foreign body he was seized with a fit of coughing, or whether the mishap was accompanied by suffocation. If so, it is well for the physician to be on his guard, as the foreign body has probably passed into the air passages, and, however small its size, cannot be spontaneously eliminated, as is the case with the digestive tract.

The patient complains of a stitch in the side that varies according to the direction taken by the foreign body. He may speak of a steady pain in the thorax, but it is especially the attacks of suffocation, dyspnœa, and coughing, that attract the attention to the air passages.

The nature of the expectoration may sometimes give information, especially with organic foreign bodies, such as date stones or beans. Several of my patients had purulent expectoration. With two others there was hæmoptysis, and with two more patients a diagnosis of phthisis had been made, whereas the symptoms were merely due to pieces of bone that were easily removed.

On examining the thorax there was with five of my patients, and especially when the foreign bodies had a certain size, an absence of motion of the ribs with diminished thoracic expansion, mensuration giving smaller figures on the side where the bronchial tube was obstructed.

Auscultation often supplies positive signs. When the foreign body does not completely obstruct the bronchial tube there is a blowing sound at the point where the foreign body is lodged. If the foreign body is large there is decreased vesicular murmur in the lobe of the obstructed bronchus, or else, if some time has elapsed since the accident, there may be accumulation of muco-pus within the bronchi, with sub-crepitant râles.

Percussion gives dulness or decreased sonority, in cases where the foreign body obstructs the bronchi.

It should, however, be clearly understood that these clinical signs only exist when the foreign body presents a certain volume. If it is small and causes little obstruction of the bronchial tube it may give rise to very few functional signs. It soon settles into place on account of its uneven surface and of the swelling and granulation of the bronchial walls and tolerance quickly becomes established. The attacks of coughing and dyspnœa, mentioned above, are caused by the change of position of the foreign body. The physical signs have, likewise, nothing characteristic when the foreign body is small, amounting perhaps at the most to symptoms of bronchitis with varying moist sounds.

Later on, when the more serious symptoms of bronchopneumonia, pulmonary gangrene or abscess of the lung occur, which may not take place for several months or even years, the diagnosis of foreign body will no longer seem probable but will be entirely thrown into the shade by the importance of the immediate condition.

The use of the X-ray is of course of great help, but the results that they give are, as was the case with foreign bodies of the œsophagus, fairly uncertain and without value, unless they are positive. In my nineteen cases only ten gave a distinct shadow on the screen. Furthermore, the results of radiography may also lead the physician astray. Thus, guided by a radioscopic shadow I have looked in the large bronchus or in the secondary bronchi for foreign bodies that did not exist. I once looked for a prune stone that

seemed to be in the right bronchus of a patient, but found nothing whatever. The same thing occurred with a pencil end in a little patient, and ulterior events showed that the bronchoscopic examination gave a correct result.

A question that seems *a priori* without interest, but which the physician has always to decide, is whether the case is one of a foreign body of the œsophagus or of the air passages. It would seem as if this diagnosis ought to be easy, and yet this is far from being the case. In the first place, the patient, who is either brought by his parents or sent by his physician, presents himself before us with the diagnosis of "oral foreign body," and rarely makes use of any other expression. It is then necessary to get him to give an exact account of the accident, which, as was said above, has, in my opinion, the very greatest value. We must inquire whether there was dyspnœa or fits of coughing, as later on the air passages become tolerant and the physician may incline rather to the diagnosis of foreign body of the digestive tract. It should, however, be remembered that a foreign body in the œsophagus, when in the upper portion of that tube, may give rise to symptoms that are entirely deceptive and lead to a diagnosis of foreign body of the trachea. I have seen this with three children, of whom two had coins in the œsophagus and the other a safety pin in the same region. They all suffered from attacks of dyspnœa, cough and suffocation, that might have led to a diagnosis of foreign body of the air passages. On the other hand, an X-ray examination was unable to settle the question, so that in the safety-pin case trachæotomy was performed to relieve the symptoms, and an attempt was made to find the foreign body with a forceps through the tracheal wound. The surgeon's astonishment was great when the foreign body was eliminated a few days later by the digestive tract.

It is evident, therefore, that foreign bodies of the air passages must quite often pass undetected, whereas on the other hand the physician quite often looks for them by mistake, guided by the clinical signs. The latency of foreign bodies of the bronchi may mislead all clinical exploration. A very good article appeared in the medical press two years ago in which several postmortems were reported where foreign bodies were found to every one's

amazement to have been the initial cause of the diverse pulmonary or pleural complaints to which the patients had succumbed.

I have been called on twice to remove a bone from a patient who had been treated for months for phthisis; the complex of symptoms was complete, with hæmoptysis, local physical signs and loss of weight. It is probable that many cases of purulent pleurisy and bronchopneumonia have a similar origin, particularly in children, who are not able to describe their sensations, and with whom the initial accident may quite well have occurred without any one being aware of it. In the present condition of our knowledge there is only one means that enables us to settle the question absolutely whether there is a foreign body of the œsophagus or of the air passages,—direct examination by means of the broncho-œsophagoscope.

With experience and skill it is always possible to perform upper bronchoscopy through the glottis without any operative wound, and to examine the trachea and bronchi or the whole of the œsophagus. This can be almost always done with grown persons with the help of cocaine; with children chloroform has to be used. This exploration is quickly carried out and is altogether harmless. I have never seen the slightest undesirable symptom nor rise of temperature after even a prolonged bronchial examination. Strict asepsis has naturally to be employed.

Exploration of the œsophagus in particular enables us to decide whether or not there is a foreign body, and to localize it with the greatest degree of precision. No other means of investigation can supply us with such certain information. We all know how easy it is for a foreign body to move down the œsophagus; an examination made even a short time after a séance of X-rays will sometimes be too late and the foreign body will have passed into the stomach. In a few hours a foreign body will in this way have disappeared and a fresh examination will show it either in the stomach or in one of the iliac fossæ. At other times the foreign body is seen by the X-rays in the upper portion of the œsophagus, but is found in the lower portion when the œsophagus is examined. From these facts it is easy to infer the mistakes that would arise if surgical intervention were the only means at our disposal for these cases. But not only is direct examination of the bronchi

and œsophagus useful from a diagnostic point of view, but is also the most exact method for the treatment and removal of foreign bodies.

With a foreign body of the œsophagus the intervention is useful to the patients in several ways. The chief element that prevents the foreign body from going down, is the spasm, the muscular contraction that fixes the foreign body in place on account of the irritation produced by its presence in the œsophagus. If this spasm is overcome the most important part of the removal of the foreign body will have been accomplished, as it will have been rendered movable. Now it is quite easy on examining through the tube to apply cocaine to the exact point where the foreign body is lodged; the circular fibres then relax and set the foreign body free. Then one of two things takes place: either it is seen to disappear into the stomach, or it can be seized with the forceps and extracted. In the case of a button lodged in the œsophagus I distinctly saw the peristaltic swallowing movements of the œsophagus take place before my eyes, but of 42 cases of foreign bodies seen and set free in the œsophagus, I withdrew only 19 with the forceps; the others went on down and were eliminated by the natural passages, which is a most favorable solution of the difficulty.

As regards bronchial foreign bodies, the treatment by means of the bronchoscope is also excellent, since out of 20 cases that I have seen, extraction succeeded in 17, 12 directly through the glottis. One of them was a failure on account of the lack of confidence of the parents which prevented my carrying my examination to its completion. In one case the foreign body was permanently fixed,—it was a pin that had passed through the bronchial wall.

These foreign bodies have a great variety of shapes. Some are round, with smooth edges, such as coins and articles of that sort; others again are very irregular, with sharp points, as is the case with bones or with artificial plates for false teeth. Difficulty in extraction may arise solely from the size of the foreign body, but it is much more apt to depend on the irregular shape, angles and corners, catching in the sides of the natural passages. Some foreign bodies have been divided *in situ,* either by means of the galvanic wire, as was the case with a dental plate in ebony, or with appropriate cutting instruments in the case of

bones. In certain cases actual *version* can be performed, after freeing the foreign body and pushing it into the widest part of the œsophagus where its points no longer catch. In four cases there was complete blocking up of the œsophagus in patients who had been examined a few days before by X-rays. The bismuth administered at that time had entirely obstructed what remained of the opening of the œsophagus in patients suffering from incomplete stenosis,—three due to epithelioma and one to spasm of the cardia. This is an accident that should be remembered; it may possibly be desirable to follow every examination under bismuth by a washing out of the œsophagus.

Œsophagoscopy also enables the physician to diagnose the lesions and complications due to the presence of foreign bodies. I have thus treated three cases of gangrenous abscess of the wall of the œsophagus due to fragments of bone or to fish bones. I succeeded through the endoscope not only in withdrawing the foreign body, but in opening and draining the abscess through the natural passages by enlarging by means of appropriate instruments the wound made by the foreign body. It is probable that in these cases an operation from the outside would have been much more serious owing to the dangers of infecting the mediastinum.

Finally, and from a retrospective point of view, I have made the diagnosis of and treated three cases of cicatricial stricture due to former lesion by foreign bodies. In these three cases there was the history of a foreign body swallowed several years before, that had remained in place for several days, and then passed down spontaneously. Inflammation had probably been the consequence, with ensuing cicatricial stricture.

From the foregoing remarks it will, I think, be manifest that in the diagnosis of foreign bodies of the œsophagus and bronchi direct examination by means of appropriate tubes can supply us with a diagnosis as exact as any one need desire, and thereby enable us to carry out treatment based on thoroughly reliable data.

Pathology

DEATH DUE TO LEUKÆMIC INFILTRATION OF THE LARYNX; PRIAPISM PERSISTENT POST MORTEM, MYELOID THROMBOSES IN THE CORPORA CAVERNOSA; FATAL HEMORRHAGE FROM SPLEEN AFTER PROLONGED RADIOTHERAPY

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DEATH DUE TO LEUKÆMIC INFILTRATION OF THE LARYNX

The patient, a housewife, aged 51, came to the University Hospital for the purpose of having a tumor removed from her abdomen. Her family history was good. She was well as a child, having the usual diseases of childhood. At 26 she had an attack of erysipelas, at 31 a severe attack of quinsy and at 39 malarial fever. Her general health has been good. Menopause two years ago. Her present illness began a year ago with attacks of headache, nausea and vomiting, and general weakness. At this time a small swelling was noticed in the left side of the abdomen and this continued to enlarge until it formed a large mass filling up the left side. During the last two months of her illness she complained of difficulty in breathing and a feeling of pressure in the chest. She had frequent epistaxis, her handkerchief being constantly stained with blood. Her bowels were loose and she developed painful hemorrhoids. She had frequent night-sweats, and her ankles became œdematous.

The physical examination (Dr. Dock) on entrance showed a body moderately emaciated, with pale skin, enlarged venules over cheeks and moderate œdema of the legs. There was no glandular enlargement. The examination of lungs was negative. Blowing murmur replaced the first sound in the left second intercostal space.

First sound at apex was murmurish. Pulse regular, quick, moderately full and strong. The left side of the abdomen contained a large, irregular, hard tumor-mass extending to the ilium. Over the lower portion of this mass the colon could be outlined by percussion. In the anterior edge of this mass two distinct notches could be felt. The lower edge of the liver extended four inches below the edge of the ribs.

The blood examination gave the following:

Red cells	2,381,500.
White cells	475,000.
Hæmoglobin	30 per cent.
Specific Gravity	1049.

Differential count (500 cells):

Small lymphocytes	.74 per cent.
Large lymphocytes	.93 per cent.
Transitional	4.00 per cent.
Polynuclears	40.70 per cent.
Eosinophile	8.00 per cent.
Mononuclears	2.20 per cent.
Myelocytes	35.00 per cent.
Degenerates	2.60 per cent.
Normoblasts	1.1 per cent.
Megaloblasts	.93 per cent.
Undetermined	4.40 per cent.

The urine examination was negative. No albumin was present.

Clinical Diagnosis.—Myeloid leukæmia. A week after admission patient's voice became hoarse; and she quickly became unable to speak above a whisper. She complained of slight pain in right side of throat in the region of the larynx; there was slight cough with little expectoration; but breathing gradually became so difficult that she was referred to the laryngologist for examination. The upper part of the larynx, particularly over the arytenoids, was found to be greatly swollen. A diagnosis of œdema of the larynx was given, and the mucosa was scarified on both sides of the glottis, which gave no relief. Some thin blood was coughed up, but no fluid came from the scarifications and the swelling did not diminish. During the day and night following, the difficulty in breathing increased and the patient complained of much soreness

in the throat. At 8.30 the next morning she died suddenly from suffocation. To the clinical diagnosis was added œdema of larynx as the immediate cause of death.

The essential features of the autopsy protocol are: Body showed moderate emaciation; skin pale; well marked œdema over lower extremities, slight over trunk and face; pale hypostasis over back; abdomen greatly distended; examination of brain negative; no hemorrhages found in any part of brain; no remains of thymus; lungs œdematous; heart dilated; valves negative; large pale clots in auricles; bronchial glands enlarged, moderately pigmented.

The abdominal tumor was the spleen extending two hand-breadths below the edge of the ribs, completely filling up the left side of the abdomen, adherent above to the diaphragm and parietal peritoneum by firm adhesions. The entire surface of the organ was covered with stringy adhesions. The upper pole of the organ was curved posteriorly, the lower pole anteriorly. Two finger-breadths above the lower pole there was a broad shallow depression in which lay the splenic flexure of the colon. In the anterior edge there were several notches; one at the beginning of the upper third was very deep, extending entirely across the anterior surface, and half way through the organ. Spleen weighed five pounds and measured 29 x 14 x 7 cm. Adrenals were negative.

Both kidneys were enlarged, firm and slightly granular. Numerous small whitish areas size of pin-point to large pin-head were scattered throughout the cortex of the left one. The liver extended a hand-breadth below the edge of the ribs in the right nipple line; was enormously enlarged, the enlargement involving chiefly the right lobe; capsule was slightly thickened; color yellowish; lobules enlarged; no lymphomata seen. Pancreas showed fatty atrophy. Right inguinal hernia admitting loop of small intestine 59 cm. long. Stomach and intestines negative. Hemorrhoids. Mesenteric and retroperitoneal glands moderately enlarged. On section they were pale, homogeneous and of soft consistency. Bone-marrow myeloid. Genital tract atrophic. Small parovarian cysts; otherwise negative. Small recent blood-clot in left broad ligament.

The *especial examination* of the upper respiratory tract showed a short, thick, rounded uvula; mucosa of the pharynx was much swollen but not œdematous; epiglottis short, very slightly recurved,

and swollen; both tonsils were large, but there was no nodular enlargement of the lymphoid tissue of the pharynx. In the right tonsil were numerous small crypts filled with calcareous granules. The mucosa of the larynx, particularly over the vocal cords and the arytenoids, was swollen so that the glottis remained almost closed; the mucosa was not translucent or wrinkled and did not present the usual appearances of œdema of the larynx; the swelling was somewhat greater on the right side than on the left. The scarifications were still visible, and the superficial mucosa showed numerous small erosions or ulcerations. Cervical lymph-glands were only slightly enlarged, and on section were soft, pale and homogeneous. Mucosa of trachea somewhat thickened. Thyroid negative.

The *microscopical examination* of the tissues and organs obtained from this case showed the usual picture of a myelæmia. The spleen, lymph-nodes and bone-marrow presented a lymphoid and myeloid hyperplasia, associated in the spleen with a marked fibroid hyperplasia. The blood-vessels of all organs were enormously distended and filled with white cells, myelocytes being in excess. The islands of Glisson in the liver showed marked leukæmic infiltration. The small white areas in the cortex of the left kidney proved to be small lymphomata containing bone-marrow cells. The blood-sinuses of the retroperitoneal hæmolymph-nodes contained great numbers of giant-cells resembling those of the bone-marrow. Nucleated red cells and mononuclear eosinophiles were also present in numbers in the spleen and hæmolymph-nodes.

The microscopical examination of sections of the mucosa of the larynx taken at the level of the vocal cords showed the blood-vessels to be enormously distended and filled with white cells, while a similar infiltration of the connective tissue of the mucosa with leucocytes explained the great swelling of the mucosa. The surface of the mucosa showed patches of desquamation and necrosis, the necrosis extending into the masses of white cells. At the surface of the infiltrations the polynuclear cells predominated, but in the deeper portions the myelocytes were in excess. The infiltration extended deep into the laryngeal muscles. Red cells were present in portions of the infiltration, and in these areas the differential count approximated that in the blood-vessels. Mitoses were found

in the cells of the infiltration, but they were also present in the circulating blood. Slight œdema of the neighboring laryngeal tissues was present.

The pathological study of this case leads, therefore, to a diagnosis of *Myelœmia with Death Due to Stenosis of the Larynx Caused by Leukœmic Infiltration of the Laryngeal Mucosa.* This infiltration is in part, at least, the result of hemorrhage into the mucosa, with subsequent local increase of the white cells through diapedesis or cell-division. It is impossible to say how much of the latter process actually occurred. In all respects this laryngeal infiltration is like that occasionally found in the brain, ear, orbit, retina, alimentary tract, etc. The hemorrhagic collections of white cells about the cerebral vessels may be so extensive as to be regarded as lymphomata. In the mucosa of the gastro-intestinal tract and bladder I have seen similar ulcerating infiltrations in myelæmia.

This case represents, I believe, the first one in which a fatal laryngeal stenosis due to leukæmic infiltrations of the mucosa of the upper respiratory tract has been observed in myelæmia. Ulcerating infiltrations have, however, been observed in chronic lymphatic leukæmia.

Pinkus, in his article on chronic lymphatic leukæmia in Nothnagel's system, says, " Leukæmic infiltrations of larynx and trachea may appear either as nodules in the mucosa with infective-traumatic ulcerations, or as diffuse, flat, slightly nodular infiltrations of the vocal cords, and the deeper parts of the epiglottis and vocal-cord region. Frequently the ulceration from these may extend into the pharynx causing local defects. Extensive necrosis of cartilage may occur. Hemorrhages are frequent but œdema of the glottis has not yet been observed." The remaining portion of the respiratory tract is rarely affected.

The leukæmic hyperplasias of the lymphoid tissue in lymphatic leukæmia and in aleukæmic lymphocytoma may cause such infiltrations and ulcerations in any body surface. They are especially common in the mucosa of the mouth and nose, and in the intestine, but occur also in the skin. In lymphatic leukæmia such infiltrations always arise in pre-existing lymphoid tissue. In the case described above the enlargement of the lymph-node was but mod-

erate. Small lymphomata were found in one kidney alone, and the pharyngeal mucosa showed no nodular enlargement of the lymphoid tissue. The infiltration of the larynx was diffuse and flat. The character of the infiltration is exactly similar to that of the orbital infiltrations causing exophthalmos and to the cerebral, retinal and auditory infiltrations that have been described as occurring in myeloid leukæmia.

The failure of the text-books to mention the possibility of such a fatal laryngeal stenosis in myelæmia must be taken as evidence of the rarity of such an occurrence. Cabot, in his article in Osler's "Modern Medicine," does not mention the possibility of such leukæmic infiltration of the respiratory mucosa in either lymphæmia or myelæmia. A few cases of leukæmia are mentioned in the literature as dying of "œdema of the glottis." Such cases may well have been identified with the one described here. Lazarus, in his article on myeloid leukæmia in the Nothnagel system, quotes Laache (Ebstein and Schwalbe's Handbuch) as an authority for the statement that laryngeal stenosis and difficulty in swallowing may be caused in myeloid leukæmia by leukæmiac infiltrations. Laache in turn refers us to Ebstein (Deutsch. Archiv, f. klin. Med., xliv, 343) as reporting a case of laryngeal stenosis caused by leukæmic infiltration. Ebstein's case is one of *acute leukæmia* (lymphatic), and the laryngeal condition was described as œdema and superficial necrosis on the epiglottis and ligamentum: aryepiglottis.

In conclusion, leukæmic hemorrhage and infiltration of the laryngeal mucosa may occur in myelæmia, causing stenosis and a clinical picture of œdema of the glottis. Such a condition should always be suspected when symptoms of laryngeal stenosis arise in a leukæmic patient. The pathological condition is identical with the leukæmic hemorrhages or infiltrations of the orbit causing exophthalmos, with those of the ear causing deafness, and with the leukæmic infiltrations of the retina, brain, etc. As to treatment the immediate fatal termination may be avoided by intubation or tracheotomy, while the use of the Röntgen rays might lessen the size of the infiltration or even cause it to disappear. Such therapeutic attempts may add days, weeks or even a few months to the patient's life.

PRIAPISM PERSISTENT POST MORTEM—MYELOID THROMBOSIS IN CORPORA CAVERNOSA

Since 1900 this laboratory has seen three cases of the curious phenomenon of priapism persisting post mortem. All three cases were advanced myeloid leukæmia from the medical clinic of the University Hospital.

Case I.—German laborer, aged 61 years, with a history of illness lasting between two and three years, beginning with general weakness, development of a tumor in the left side and finally severe diarrhœa. For four months his penis remained persistently in a state of nearly complete erection. The glans was exposed and the foreskin completely retracted. The erection involved chiefly the corpora cavernosa. There was no pain, but entire absence of sexual feeling. Urination was difficult. Blood showed 750,000 white cells, 2,250,000 reds, hæmoglobin 40 to 50 per cent. About 44 per cent. of the white cells were myelocytes. Patient died during an acute severe diarrhœa.

The most striking thing seen in the inspection of the cadaver was the very long and large penis with large glans exposed and foreskin retracted. The organ projected downwards slightly above the level of the thighs. The corpora cavernosa were fairly firm and hard, while the glans and the corpus spongiosum were softer. The tonsils were enlarged, and the mucosa of the larynx swollen. The spleen weighed six pounds nine and one-half ounces. Numerous small lymphomata found in cortex of both kidneys. Mucosa of colon swollen and injected. Lymph follicles enlarged. Liver greatly enlarged and contained numerous small lymphomata. Retroperitoneal glands enlarged and pale. Aorta sclerotic. Bone-marrow hyperplastic. Section of the penis showed spaces of the corpora cavernosa to be filled with small pale friable thrombi. No microscopical examination was made of the penis.

Diagnosis.—Leukæmic thrombosis of corpora cavernosa.

Case II.—American, clerk by occupation, aged 27. History of illness lasting about a year. Symptoms of general weakness developing rapidly. Large tumor in splenic region. Priapism for four weeks. After marked mental disturbances lasting for several days became comatose, remaining so until death. Leucocytes just

before death were 900,000, about 40 per cent. being myelocytes. Numerous cells of the type of large lymphocytes were also present. On inspection of the cadaver the large swollen penis with exposed glans and retracted foreskin at once attracted attention. Instead of dropping down between the thighs the organ projects downward and slightly upward above the horizontal plane of the body. It is freely moveable and when laid back upon the abdomen reaches to the navel. The cadaver is that of a well-built and well nourished young man. Numerous small hemorrhages were found throughout the brain. Mucosa of larynx swollen. Heart dilated, filled with pale plum-colored blood having the consistency of a thick pea-soup. Would spread with a knife like soft butter. No clots in heart. All vessels enormously dilated and contained the same thick plum-colored blood. Spleen greatly enlarged, reaching within a hand-breadth of pubis and a finger-breadth to the right of the median line. Measured 30 x 20 x 10 cm. and weighed 5180 Gm. Liver also greatly enlarged, weighing 6144 Gm. Mesenteric and retro-peritoneal glands enlarged, very soft. Section of penis showed corpora cavernosa to be filled with thick, pale, plum-colored blood containing small masses of agglutinated leucocytes. All other organs and tissues showed the appearances of a myelæmia with very high white-cell count and marked hæmolysis of the red cells.

Case III.—American, aged 49. Case of chronic myelæmia. Prolonged Röntgen ray treatment. (History given below. Same case as that dying from hemorrhage from spleen.) Eighteen months of Röntgen ray and arsenic treatment caused great improvement. Relapse occurred and after a short period of gradually increasing weakness and excessive sweating death took place. During the last seven months of his life he had had a constant full erection of his penis; sexual feeling lost; no pain; urination difficult. Entire organ removed at autopsy and prepared for microscopical study. All other organs showed changes characteristic of chronic myelæmia.

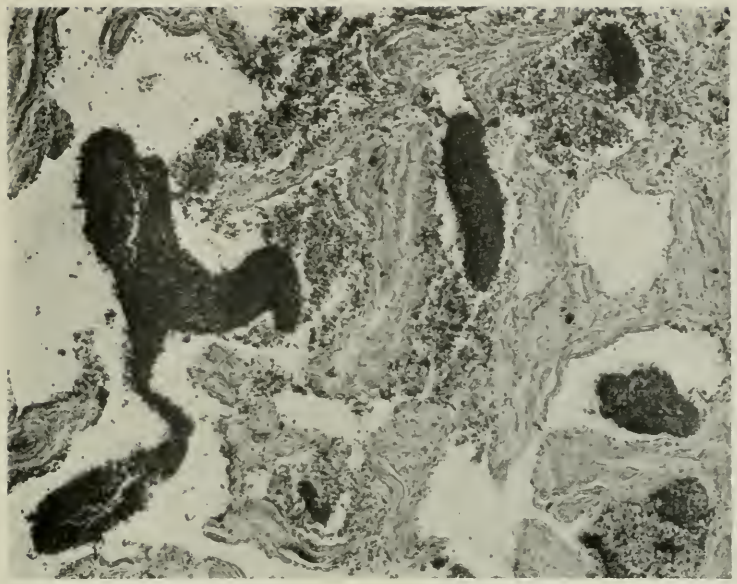
The corpora cavernosa presented greatly dilated blood-spaces; those of the corpus spongiosum were moderately dilated. In these blood-spaces there was present a large amount of fresh unclotted blood showing an enormous preponderance of leucocytes. Myelocytes were in the great majority. In nearly every blood-space of the corpora cavernosa there was present beside the fresh blood a

deeper stained, more compact, mass of leucocytes and red cells. In these masses the leucocytes which were mostly mononuclear and of the myelocyte type were crowded closely together. In some spaces it was difficult to make out the individual white cells. They were contracted and stained very deeply (pyknotic nuclei); or showed karyorrhexis and karyolysis. The red cells between the leucocytes were fused and agglutinated together into irregular masses still containing hæmoglobin. Very little fibrin was present in these masses. In some of the larger masses the cells stained well, their nuclei were large and bright, no granules could be found in the cell protoplasm, and the white cells resembled large lymphocytes rather than myelocytes (Figs. 1 and 2). Mitoses occurred in these areas, and the red cells had disappeared. Such collections of cells resembled the bone-marrow of the patient and suggested a proliferation of marrow-cells (myelocytes) within the blood-spaces of the penis. The majority of the cell-masses must, however, be regarded as agglutination-leucocyte and red-blood-cell-thrombi of different ages as shown by the various stages of degeneration and necrosis. No organization of any of these thrombi was seen. The muscle-coats of the blood-vessels and the tissue of the trabeculæ showed no leucocyte infiltration. The walls of the blood-vessels of the penis were sclerotic and showed more or less hyaline change.

In these three cases we see examples of prolonged priapism due to leukæmic stasis and thrombosis of the blood-spaces of the penis. The solid, contracted masses of agglutinated cells in the cavernous spaces explain fully the phenomenon of continued erection and its persistence after death. To what extent there was a new formation of myelocytes in these thrombi cannot be decided from the sections. Could it be shown to occur to any great extent the masses of cells would have to be regarded as growing metastases and not as simple thrombi.

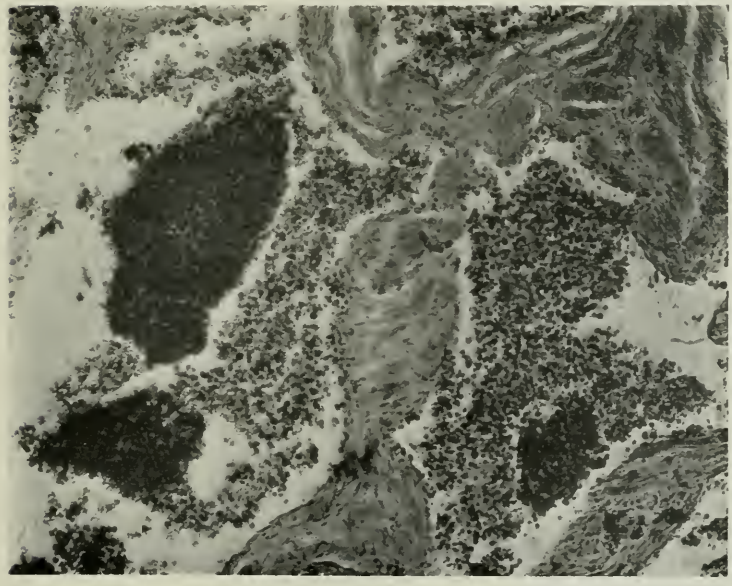
Prolonged priapism is of rare occurrence and has been described in the literature relatively seldom. Terrier and Dujarier in 1907 could collect only 48 cases. The cause of the priapism in these reported cases was regarded as nervous, traumatic, inflammatory, leukæmic or idiopathic. Metastases of malignant tumors into the penis may also cause priapism. Of the 48 cases 16 were leukæmic. Leukæmia is, therefore, the most common cause of priapism. More-

Fig. 1.



Myeloid thromboses in corpora cavernosa; priapism persistent seven months.

Fig. 2.



Higher power view of myeloid agglutination; thrombi in corpus cavernosum, leukæmic priapism.

over, since the priapism may appear as a precursor of leukæmia, it is not improbable that some of the cases of priapism regarded as idiopathic were also leukæmic. A history of priapism was obtained in about one-fourth of our autopsy cases of leukæmia, so that its occurrence in this disease cannot be regarded as rare as many authors would have it. Nevertheless, Cabot in Osler's "Modern Medicine" says it was not observed in any of the 89 cases of his series. Unless especial effort is made to obtain such information from the patient, knowledge on this point may not be forthcoming. In the clinical notes of our three cases no statement occurred as to the condition of the genital organs, and the duration of the priapism was learned only after the autopsy by inquiries of house physician, internes and clinical assistants.

As to the nature of leukæmic priapism various opinions have been held. Three chief theories are given in the text-books: that of the nervous origin of the priapism, thrombosis, and the theory of a hæmatoma or neoplasm preventing the return of the venous blood. The nervous theory has been largely accepted in this country as an explanation of any form of priapism and the leukæmic cases have been referred by many authors to this category. Support for this view has been found in the fact that priapism occurs occasionally after fractures or dislocations of the vertebræ, particularly of the cervical or upper dorsal region. For the majority of cases of persistent priapism some other cause must be sought. Even in fracture of the spinal column it may be caused by something else than nervous influences, as in a case reported by Tauzki, in which the priapism persisted thirty-six hours after death. Such a phenomenon would indicate the action of other factors than nervous impulses. Only in cases where there is a distinct medullary lesion can the nervous origin of priapism be asserted with any degree of certainty.

The obstruction to the return of the venous blood by a malignant tumor or hæmatoma no doubt explains a certain number of cases. Vorster saw a hæmatoma of the corpus cavernosum compressing the urethra. In traumatic cases such an origin must be borne in mind. In leukæmic cases associated with hemorrhagic tendency such a hemorrhage into the penis might occur, particularly when conditions, such as trauma, alcoholism, excessive and violent attempts at

intercourse, aided. In extensive metastasis of malignant tumors the involvement of the blood-spaces of the penis may take place, although rarely. Persistent priapism due to this cause has been recorded, and the writer has seen one case in which the corpora cavernosa were filled with carcinoma metastases. Persistent priapism had occurred before death: post mortem the size of the penis diminished, the organ remaining in a state of incomplete erection.

Insofar as the leukæmic cases are concerned the theory of thrombosis or blood-stasis has recently gained much favor, and most of the recent observations upon this subject express this view. There have been, however, only a few autopsy examinations of the penis in cases of persistent priapism. Terrier and Dujarier were able to collect only four autopsy cases: Jadioux, probably the first instance (1845) observed in a leukæmic case; Kanders, a case of miliary tuberculosis; Kast, a case of leukæmia; Tauzki, a traumatic case. Of these cases only two are described sufficiently in detail to give us any definite clue as to the pathogenesis. Kast and Kanders both found distinct evidences of thrombosis. In Kast's case the thrombi had undergone organization, the corpora cavernosa being converted into firm fibrous tissue, while the corpus spongiosum appeared dilated and normal.

Surgical operation has been carried out in ten cases and in these the operators found the corpora cavernosa filled with a black, syrupy blood flowing very slowly or not at all, rarely with small clots. The results of such surgical examinations favor, rather than otherwise, the theory of thrombosis as the cause of the priapism in the majority of cases. They confirm the existence of a venous stasis. Since Terrier's and Dujarier's paper I have found four cases of leukæmic priapism not mentioned by them (Blum, 1906; Gunckel, 1906; Kunst, 1907; Favera, 1908). Blum believed a soft thrombus to be the cause in his case; Gunckel inclines to same view; and Favera definitely states that leukæmic priapism is the result of a marked venous stasis due to obstructed venous outflow from the corpora cavernosa caused by thrombosis of the latter.

The case reported by Kunst (Med. Klin., 1907) offers especially interesting features. Support of the splenic tumor by a supporting bandage caused a lessening of the pains and a complete disappearance of the priapism from which this patient suffered. The hyper-

æsthesia of the anal region, scrotum and penis with exception of glans and prepuce, pointed to an irritation of the 3–5 sacral segments leading to sympathetic disturbances. Kunst assumes a damage to the sympathetic from the spleen tumor leading to a vasomotor priapism. This would cause only a soft swelling of the corpora cavernosa; for the complete erection the irritation of the sacral centre of the Musc. perinei prof. through reflex impulses would be necessary. While we cannot wholly exclude a nervous pathogenesis for some of these cases, yet Kunst's case can be easily explained as due to a temporary stasis or thrombus.

The cases from the literature and my three autopsy cases support the view that leukæmic priapism is the result of a leucocyte stasis and agglutination-thrombosis of the spaces of the corpora cavernosa. Such an agglutination may be temporary and the priapism may be short or prolonged over many months. It is especially likely to occur when the leucocyte count is high and when there is a high degree of hæmolysis and leucolysis. The tendency of the leucocytes to stick together or to form balls in the circulating blood in leukæmia is often seen in the heart and large veins. Such an increased viscosity of the white cells may aid in the formation of such myelocyte thrombi. The absence of fibrin, the failure of attachment to the wall with organization, the preservation of the endothelium, etc., all point to the changes in the blood itself as the essential cause of the clumping. Degeneration or proliferation of the atypical white cells in the masses will follow, the first-named process being the most common.

In regard to inflammatory and infective processes in the penis causing priapism it is not necessary to take up here that feature of the subject. Syphilis, chancroid, gonorrhœa, tuberculosis and pyogenic infections may all cause local conditions favoring thrombosis of the cavernosa or spongiosum (cavernitis). Such secondary inflammatory priapism may be coincident with leukæmia.

As to the post-mortem persistence of priapism it has been observed in trauma, fractures of vertebræ, death by hanging, and a few other conditions. My cases are, I believe, the first ones observed in leukæmia.

The symptoms of leukæmic priapism vary. The duration may be short or long, attacks single or repeated, painful or painless,

usually the latter. Sexual feeling is lost and emissions usually do not occur. Urination is more or less difficult. The psychical effect is bad, and the three patients described above were ashamed and greatly depressed over the condition. Long-continued attacks are likely to occur just before death, and in this way the condition becomes of prognostic import. It may, however, be a very early sign of the disease; and it is important to examine carefully the blood in all cases of idiopathic priapism.

Treatment should be carried out, if simply for the psychical effect. The use of Röntgen rays directly on the penis would theoretically appear to be the most rational thing to do. By the destruction of the agglutinated white cells the cause of the obstruction and stasis would be removed. Blum also advises this treatment and quotes a case of Goebel's in which local irradiation produced an immediate cure of the priapism. Radiotherapy failing, surgical operation might be performed, as surgical measures have resulted in a cure in other forms of obstructive priapism.

FATAL HEMORRHAGE FROM SPLEEN FOLLOWING RADIOTHERAPY

The following case presents several features of especial interest. In addition to a priapism persistent post mortem, this patient (Case III, above) had received a long course of radiotherapy, the results of this treatment being reported by Roth (Jour. Amer. Med. Assoc., 1906).

Mr. J. D., American, aged 49, was admitted to Dr. Dock's clinic in March, 1905. His chief symptoms at that time were a dull, heavy feeling in the stomach region, vague abdominal pains, distress after eating and general weakness. On examination he was found to have a greatly enlarged spleen and the blood examination was as follows:

Red blood-cells	2,500,000.
Leucocytes	135,000.
Hæmoglobin	70. per cent.
Small lymphocytes	2.0 per cent.
Large lymphocytes	0.4 per cent.
Transitionals	2.2 per cent.
Polynuclears	42.6 per cent.
Eosinophile polynuclears	.2 per cent.

Eosinophile mononuclears	.8 per cent.
Myelocytes	45.6 per cent.
Mast cells	5.4 per cent.
Degenerates	0.6 per cent.

Röntgen irradiation was carried out; at first over the splenic region alone, later over spleen and long bones alternately, in exposures of 7–15 minutes, the total number of exposures equalling 2800 minutes. At first the white count rose, reaching a maximum of 365,000 in one month; after three months returned to 135,000 and then gradually fell during the remainder of the year. Arsenic was then given in connection with the radiotherapy and in two months the leucocytes fell to 4680, remaining at a normal level at the time of Dr. Roth's report. Patient left hospital April 13, 1906, feeling very well. During the summer he felt fairly well except that about every two weeks he had attacks of weakness and general distress. At times vomited and belched gas and was constipated. During these attacks he went to bed, although during the remaining time he was able to be out of doors. In August he began to be worse and to have severe pain in the splenic region. Priapism began before leaving hospital and lasted all summer. Returned to hospital because of these symptoms. During the summer the radiotherapy was discontinued but the arsenic was taken at regular intervals. On re-entering hospital he was found to have a temperature of 102.4 with herpes on nose and lips. The entire left side of the abdomen was filled with a splenic tumor reaching from edge of ribs to Poupart's ligament and extending two inches to right of the median line; œdema over spleen; abdomen tender; superficial glands not enlarged; slight œdema over ankles. The blood examination was as follows:

Red blood-cells	3,000,000.
Leucocytes	256,000.
Hæmoglobin	75–80 per cent.
Myelocytes	65.3 per cent.
Polynuclears	17.0 per cent.
Mast cells	11.0 per cent.
Transitional	4.6 per cent.
Lymphocytes	1.3 per cent.
Polynuclear Eosinophiles	.5 per cent.
Mononuclear Eosinophiles	.3 per cent.

Iron (Blaud's pills) and Fowler's solution were ordered and irradiation again advised. One exposure only was given. Complained of great pain in splenic region, moaned constantly in sleep, pulse very feeble, 110–130, temperature 100–101°. Constant sweating. Quickly became very weak. Symptoms of collapse, fell into a stupor, and died on the third day after admission.

Autopsy revealed the condition of priapism described above. On opening the abdomen the left side was found to be filled with the greatly enlarged spleen, the upper half of which was embedded in a fresh blood clot. There were numerous adhesions between the anterior surface of the spleen and the abdominal wall. The left half of the diaphragm was pushed high up and between it and the upper pole of the spleen there was a large laminated blood-clot of the size of a child's head. The outer layers of this clot were brownish in color, dry and friable, the inner mass consisted of a recent clot and some fluid blood. On carefully removing the clot from the surface of the spleen, a funnel-shaped opening the size of a lead-pencil was found through the capsule of the anterior surface near the beginning of the upper third. From the opening a small amount of blood exuded on pressure. The clot about the opening was more firmly adherent and showed evidences of beginning organization. Exploration of the opening showed an irregular tortuous space beneath the capsule extending into a greatly dilated splenic vein.

The surface of the spleen was mottled, pale-yellow areas surrounded by reddish streaks (infarcts). The opening into the splenic vessel occurred just at the border of one of these infarcts. The greater part of the splenic tissue appeared to be necrotic (infarcted). No large thrombi were found in the splenic vessels, but the smaller vessels were for the greater part thrombosed. All other organs and tissues showed the characteristics of advanced myeloid leukæmia (ecchymoses in pericardium, etc., enlarged liver, small areas of leukæmic infiltration in kidney, etc.). The spleen, with clot removed, weighed 3000 Gm., and measured 32 x 19 x 11 cm. Liver weighed 2200 Gm. Retroperitoneal lymph-nodes and hæmolymph-nodes all enlarged.

The microscopical examination showed characteristic changes of advanced myelæmia with extensive anæmic infarcts and hemor-

rhages in spleen. The organ was markedly fibroid and there was but little living lymphoid or myeloid tissue left. Numerous arteries showed an obliterating endarteritis with thrombosis. The veins were greatly dilated and in part thrombosed. Hemorrhage by rhexis from large vein communicating with opening through capsule.

Diagnosis.—Chronic myelæmia with fatal hemorrhage from spleen. Extensive anæmic necrosis of spleen. Hemorrhage per rhexis of splenic vein. Myeloid transformation of bone-marrow and hæmolymph-nodes.

Anæmic infarction and hemorrhage are very common occurrences in the spleen of chronic myelæmia, and often attain a marked degree of size. I have not found, however, any observation recorded of a case like this with fatal hemorrhage per rhexis through the capsule into the peritoneal cavity as the result or accompaniment of an anæmic infarction. Rupture of the spleen has, however, been observed in both lymphæmia and myelæmia, and this case, no doubt, bears a very close relation to these. The extensive necrosis of the infarcts and the distended and weakened vessel formed the conditions favoring the local rupture of the capsule and the resulting hemorrhage into the peritoneal cavity. That this hemorrhage was a relatively slow one and had persisted for several days at least was shown by the older outer laminæ of the clot and the beginning organization.

That the radiotherapy had any causal relationship to the extensive vascular changes and necrosis of the spleen cannot be decided. Anæmic infarcts and hemorrhages are common enough in leukæmic spleens not irradiated; but such extensive changes as seen in this case are certainly very rare, and we cannot, therefore, escape the thought that the treatment may have had something to do with the production of such great changes. Such a possibility must be borne in mind in the radiotherapy of leukæmia.

A SOLID TERATOMA OF THE MEDIASTINUM WITH HISTORY AND AUTOPSY NOTES

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Tumors of the mediastinum are not uncommonly met with but the one here reported is of unusual interest because it presents a great complexity of structure and contains tissues derived from all three germ layers of the embryo.

Tumors of this kind have been collected and reviewed by Christian [1] who divides them on the basis of their structure into three groups as follows:

1. Cysts of slight complexity; essentially dermoid cysts of ectodermal origin.

2. Tumors of great complexity; containing derivatives of all three germ layers (teratoma).

3. Tumors of the first or second class, which, in some part of their structure, show malignant character and form metastases.

The tumor about to be described must be placed in the second group—solid teratoma—of which, so far, only six cases have been reported.

Following is the clinical history and the autopsy notes:

H. K., male, an Armenian laborer, entered the Samaritan Hospital on September 28, 1908, complaining of cough, shortness of breath, pain in the chest, particularly on the left side, and pain in the abdomen.

Nothing of his family or personal history could be obtained as he could not speak English. The only information that could be obtained relating to his illness was that he had been sick about two weeks.

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[1] Solid teratoma of the mediastinum, Jour. of Med. Research, vol. xvi, No. 2, pp. 275–286, 1907; Tumors of the mediastinum, Osler's "Modern Medicine," vol. iii, pp. 890–910.

Physical Examination.—This showed that the patient was well built and well nourished. The body was covered with a profuse perspiration. The lips were cyanotic. The pupils were equal. There was no œdema of the face or extremities.

Chest.—The respiratory movements were absent on the left side. The chest was symmetrical. The vocal fremitus was increased on the right and entirely absent over the left side. The left front was flat on percussion and in the back, flatness extended from the fourth dorsal spine to the first lumbar. On the right front an area of dulness was found beginning at the second rib 3 cm. from the sternum and extending to the upper border of the fourth rib, merging into the dulness of the left side. At the right base posteriorly there was a paravertebral triangle of dulness (Grocco's sign) 8 cm. wide extending from the tenth dorsal to the first lumbar spine. Percussion of the sternum caused considerable pain. The respiratory murmur on the right side showed a prolongation of expiration accompanied by mucous and sonorous râles. On the left side there was an entire absence of the breath sounds over the area of flatness with ægophony.

Heart.—The point of maximum impulse is seen and felt in the fifth intercostal space 3 cm. to the right of the sternum. The cardiac dulness could not be well mapped out. The heart sounds were clear and no adventitious sounds were heard. The pulse was 120 per min., regular, and full. The vessels were soft; the blood pressure 124 mm. (Riva Rocci). The temperature was 99° F.

The *spleen* was not palpable and the outline could not be made out by percussion.

The *abdomen* and abdominal organs on examination presented nothing of note.

A diagnosis of left pleural effusion with displacement of the heart and liver was made. The patient, however, did not permit the aspiration of the fluid.

October 8, 1908.—The patient has grown rapidly worse. The perspiration is profuse and continuous. The temperature has been very irregular, 96°–101° F. The pain in the head, chest, and abdomen has been more severe; the paroxysms more frequent and longer. The dyspnœa has increased. Consent was given to remove the fluid and 3 litres of blood-stained serous fluid were withdrawn. Examination of the fluid showed it to be a transudate. Enumera-

tion of the cells present after centrifuging the fluid showed the small mononuclears 27 per cent., polymorphonuclears 28 per cent.

By percussion of the chest, after the removal of the fluid, an area of dulness was revealed beginning at the second rib, 3 cm. to the right of the sternum and extending downward to the upper border of the fourth rib. To the left it arched upward to the clavicle and then downward to the upper border of the third rib just within the mammary line. A skiagraph made at this time revealed a mass corresponding in position to the dulness just noted.

The withdrawal of the fluid made the patient more comfortable for a few days; but the fluid quickly began to accumulate again, causing even greater dyspnœa than before. The cough was severe and the sputum was profuse. The tenderness of the sternum was increased.

October 19, 1909.—The patient's condition has grown continually worse. Percussion over the sternum was very painful. There was no bulging, pulsation or thrill. There was no tracheal tug. The larynx was normal. The pulse was weaker in the left radial than in the right. A pulsus paradoxus is noted in the right radial. The left side of the face and the left arm have become swollen and the veins on the left side of the neck, chest and abdomen are distended and prominent.

Clinical Diagnosis.—Mediastinal tumor causing compression of the left innominate vein. Compensatory circulation through the internal mammary and deep epigastric and ascending lumbar veins. Left pleural transudate.

Exitus.—October 29, 1908.

Autopsy Report.—Length of body 170 cm. Rigor mortis not present. The pupils are equal. The chest shows extensive dilatation of the superficial veins, more marked on the right side where they extend into the neck. The chest is full but symmetrical. The left arm is considerably larger than the right in circumference but does not pit on pressure. There is some dilatation of the veins of the abdomen, more marked on the right side. There is no œdema of the feet or ankles. The lymphatic glands in the axilla, above the clavicle and in the groin are not enlarged. When the peritoneal cavity is opened there is no escape of gas. The coils of the intestines are distended. The appendix is in the right iliac fossa and is normal.

The *liver* extends 10 cm. below the costal margin in the middle line. The diaphragm is at the sixth rib on the right side and at the seventh rib on the left side.

When the thorax is opened, the lungs do not collapse. When the sternum is removed a mass presents on the left side measuring 22 cm. in length, extending from the upper border of the sternum to the diaphragm. It is rather firm in consistency and is encapsulated. It occupies the cavity of the mediastinum and its upper surface is firmly attached to the anterior chest wall. Both pleural cavities contain a moderate amount of fluid deeply blood-stained. The left lung is displaced backward and is much compressed by the mass just mentioned.

Heart.—The pericardium contains an excessive amount of clear serous fluid. The apex is slightly to the left of the median line. The heart is pressed to the right and downward. It measures 11 x 10 x 4 cm. On section the left ventricular wall is thinner than usual and measures 1 cm. in thickness. The mitral valve is smooth but has a dark-brownish color and is blood-stained. The tricuspid valve is smooth but the orifice is dilated. The auricular appendages contain no clots and the heart muscle is soft and flabby. The pulmonary and aortic valves are normal.

Right Lung.—This measures 27 x 20 x 6 cm. and is distinctly emphysematous. It is everywhere crepitant and free from adhesions. On the under surface of the lower lobe a nodule can be felt which on section is found to be calcified and surrounded by deeply pigmented tissue. On section the lung tissue is deeply congested, especially the lower lobes, but aside from that presents no abnormality. The bronchial lymphatic glands show a few foci of caseous tuberculosis but no secondary tumor growth is found.

The *left lung* is found firmly adherent on its median surface to the surface of the tumor mass. It seems, however, to be distinct from the tumor and on section it is found to be markedly œdematous and does not crepitate. The left bronchus seems to have been pushed outward and upward by the mass which lies directly upon it. Its outline is round and does not seem to have been compressed by the tumor. The trachea, likewise, is not compressed.

The aorta runs slightly to the left of the trachea and has a normal appearance.

The *tumor mass* measures 30 x 20 x 10 cm. and weighs 4300 grammes. The growth on section appears to be very cellular and is extensively degenerated. The cut surface has a mottled reddish-white color, the light areas representing the preserved portions, and the red the degenerated portions. It is apparently without stroma and is composed almost entirely of cells of the tumor. There are numerous hemorrhages in the tumor tissue and many points of suppuration are found, probably due to secondary infection.

The *liver* measures 30 x 25 x 6 cm. The capsule is smooth, but on the outer surface, both above and beneath, are found numerous nodules measuring about 1 cm. in diameter, which project above the surface of the liver. On section the liver shows an increase in connective tissue and has a typical nutmeg appearance. The nodules referred to are found to be metastases from the main tumor mass and correspond in appearance to it.

The *spleen* measures 14 x 8 x 6 cm. The capsule is smooth and on the lower border shows four notches. On section it shows a slight congestion and increase in the connective tissue, but in other respects presents a normal appearance. The adrenals are normal.

The *left kidney* measures 12 x 7 x 5 cm. The capsule strips readily, leaving a smooth surface. The cut surface shows the markings very distinct and there seems to be a congestion about the margins of the pyramids.

The *right kidney* measures the same as the left and on section presents a similar appearance.

The *mesenteric lymphatic glands* are not enlarged.

The *pancreas* is normal in size and appearance as well as on section.

The *stomach* is distended with retained gastric secretion and the mucous membrane has a normal appearance.

Anatomic Diagnosis.—*Sarcoma* of the mediastinum; *metastases* to the liver; *calcified tuberculosis* of the right lung; *œdema* and *atalectasis* of the left lung with complete obliteration of the pleural cavity on the left side; chronic passive *congestion* of the spleen, liver, and kidneys; *enlargement* of the left arm; and *distention* of the superficial veins of the chest and abdomen.

The tissue saved for microscopic examination was hardened in Zenkers's fluid and imbedded in celloidin and paraffin. The

sections of the tumor were stained with eosin and methylene blue, hæmatoxylin and eosin, Van Gieson and Mallory's connective tissue stain.

The sections from the tumor show that only a relatively small part of the tissue is preserved, the greater portion is degenerated to such an extent that the structure cannot be made out.

In sections where the structure is still preserved it is found that the stroma is in excess of the parenchyma. It presents a varied picture. The greater part is ordinary fibrous connective tissue, but in many places it is a very loose meshwork which resembles undifferentiated mesenchyme. In other parts the connective tissue cells are more oval, the nuclei larger, and the cells closely packed together, presenting an appearance exactly like the connective tissue of the mucosa of the uterus. The irregular arrangement and the inequality in size of the cells in these areas suggest a sarcoma. A few nodules of embryonic hyaline cartilage are found imbedded in the fibrous connective from which they evidently originate. No distinct blood supply is found.

The epithelial tissue, although less in amount than the stroma, presents many different varieties. It is present for the most part as a lining to cysts and gland spaces which are very numerous. The cysts and glands are of all shapes and sizes, some with smooth lining, some with finger-like processes extending into the lumen. A distinct tunica propria is found about some of the glands. The contents are serous or mucous or composed of desquamated cells or filled with purulent exudate.

The epithelial cells are either in a single layer or stratified, mostly of low cuboidal shape, occasionally high columnar, the height of the cells being undoubtedly influenced by the pressure of the contents. Ciliated cylindrical cells and goblet cells are found lining some of the cysts. The epithelium of entodermic origin predominates. A few masses of stratified squamous epithelium, occasionally with epithelial pearls are found. There are no evidences of malignant growth seen in the epithelial structures. A few masses of what closely resembles neuroglia tissue are found generally round in shape and sharply separated from the adjacent tissue. The cells have fine, interlacing, wavy fibrils extending from the protoplasm. The nuclei are oval or round and deeply staining. No ganglion cells are found.

Liver.—The metastatic growth in the liver presents an appearance closely resembling the original tumor. The stroma of connective tissue is in excess but shows a less varied growth. It is for the most part a loose reticulum of cells. No dense fibrous tissue and no cartilage are seen. No distinct blood supply can be found. The epithelial structures are in the form of cysts and glands. The cysts are larger than in the original growth and many are filled with red blood-cells. The glands are in the form of long single or compound tubular glands. Some have a smooth, others a polypoid lining. The epithelium is of flat cuboidal, columnar and ciliated columnar cells, generally one type of cell lining a cyst or gland; occasionally several types are present in the same cyst.

The parenchyma of the liver shows no increase of connective tissue between the lobules. The central veins are congested and the liver cells near them are cloudy and stained poorly. In the outer portion of the lobules the cells show a moderate degree of fatty metamorphosis.

Right Lung.—The pleura is not thickened. The alveolæ are for the most part filled with serum and often contain large pigmented epithelial cells (Herzfehlerzellen). Many of the alveolar walls have been ruptured.

Left Lung.—The pleura is much thickened by connective tissue which is infiltrated with small mononuclear leucocytes. The alveolæ are packed with polynuclear leucocytes, plasma cells, and desquamated epithelium. Fibrin is present in some of the alveolæ. Scattered through the section in the blood-vessels and alveolæ are dark blue staining masses which are masses of bacteria.

Heart Muscle.—Shows nothing of note.

Spleen.—There is an increase in the amount of the pulp due to a congestion with red blood-cells. The central arteries in the trabeculæ are thickened and show hyaline degeneration of the walls. Masses of bacteria are scattered through the section.

Kidneys.—The capsule is not thickened. The cortex shows an extensive cloudy swelling. A few hyaline casts are found in the tubules of the medullary portion. Masses of bacteria and bacterial casts are also present.

The *adrenals, pancreas, bladder, prostate,* and *testicles* show no abnormalities to be noted.

Revised Diagnosis.—*Solid teratoma* of the *mediastinum* with metastases to the *liver; calcified tuberculosis* of the right lung, *œdema* and *atalectasis* of the left lung, with universal fibrous adhesions obliterating the entire pleural cavity; chronic passive *congestion* of the *spleen, liver* and *kidneys; enlargement* of the left arm and distention of the superficial veins of the chest and abdomen, due to compression of the left innominate vein and the formation of a compensatory circulation through the internal mammary; deep epigastric and ascending lumbar veins.

A comparison of the structure of the metastatic growths in the liver with that found in the original tumor shows that the former is much less complex. Tissue elements representing differentiated mesoblast and hypoblast are present but none originating from the epiblast can be found, although they were present in the primary growth.

The connective tissue in the metastases is of a lower degree of differentiation, nearly all of the cells being of one type. The same is true of the epithelium lining the cysts and gland spaces (hypoblast) which show much less variety than in the primary tumor. It is quite possible, however, that greater differentiation might take place with a greater growth of the metastases.

It is clear nevertheless that the metastases represent a reproduction of the primary tumor as a whole and not of any one tissue in it. This would seem to indicate that in the formation of the secondary growths from these tumors that an individual or a few cells from the primary tumor carried in the blood- or lymphatic-vessels to other organs may develop into growths of very complex structure containing tissue from all of the three germinal layers.

Such cells must possess great differentiating power, yet it seems much more likely that complex secondary growths originate in this way than that cells representing each tissue group metastasize and reproduce their kind.

If this is true of the secondary growths it is probably true of the primary growth which might take its origin in the same way from the inclosure or misplacement of undifferentiated cells from the germinal layers of the embryo. This conception of the origin of teratoma accounts for their development in any organ of the body.

INDEX TO VOLUME IV

(NINETEENTH SERIES)

A

Addison's disease, 88, 91
Adenofibroma of breast, 132
Adherent placenta, gravity method of removal of, 199
Adhesions, postoperative, 141
Adrenal extract in adrenal disease, 97
  insufficiency, 88
    acute, 93
Age factor in prognosis of advanced tuberculosis, 100
Alcohol as a food, 59
Anæmia, diagnosis and treatment of pernicious, 67
Anæsthetic, ethyl chloride as a general, 182
  in prostatic operations, 240
Angioma of face, 129
Anorexia nervosa, diet in, 57
Antimeningitis serum and the results of its employment, 1
  indications for the employment of, 7
  manner of administration of, 8
  manner of therapeutic action of, 5
  preparation and standardization of, 2
  results of, 14
Aortic regurgitation, 118
  stenosis, 118
Appendicitis, acute, 134
Appendicostomy, 82
Arsenic in pernicious anæmia, 86
Autointoxication, diet in, 58

B

Becker, E. W., and Carey, H. W., A solid teratoma of the mediastinum with history and autopsy notes, 296
Bierring, Walter L., The diagnosis and treatment of pernicious anæmia, 67
Bladder, papilloma of, 142
Blood in infantile typhoid, 242
  in leukæmia, 281
  in pernicious anæmia, 69
Bone-marrow in pernicious anæmia, 69
Brain, sarcoma of, 127
Breast, adenofibroma of, 132
  carcinoma of, 133
Bronchi and œsophagus, foreign bodies in, 272
Bronchoscopy, 278

C

Cæcum, perforation of, 134
Carcinoma of breast, 133
  of lip, squamous, 130
  of prostate, 238
  of pylorus, 138
  of uterus, early diagnosis of, 193
  treatment of, by fulguration, 22
Carey, H. W. and Becker, E. W., A solid teratoma of the mediastinum with history and autopsy notes, 296
Cholecystitis, 137
Cholelithiasis, 137
Circulation in adrenal insufficiency, 89
Colonic irrigation in pernicious anæmia, 85
Color photography, history of, 167
  in relation to surgery, 166
  technic of, 171
Congenital syphilis, 247
Corpora cavernosa, myeloid thromboses in, 280
Coryza in infants, 248
Cullen, Thomas S., The early diagnosis of cancer of the uterus; operative technic, 193
Cumston, Charles Green, Epiploitis following the radical cure of hernia, 145

D

Deaderick, William H., Hymenolepis nana in the United States with report of two cases, 252
Deaver, John B., The indications for surgery of the prostate, 228
Delusions, hypnotism in, 62
Diarrhœa in pernicious anæmia, 84
Diet, general consideration of the therapeutic uses of, 54
Digestion factor in prognosis of advanced tuberculosis, 103
  in adrenal insufficiency, 89

Dipsomania, hypnotism in, 63
Duckworth, Sir Dyce, Gouty phlebitis, 108
Dyspepsia, hypnotism in, 63

E

Endocarditis, rheumatic, 115
Eosinophiles in Hymenolepis nana, 258
Epididymitis, 240
Epilepsy, Jacksonian, 128
Epiploitis, diagnosis of, 161
following the radical cure of hernia, 145
pathology of, 150
symptoms of, 154
treatment of, 162
Ethyl chloride as a general anæsthetic, 182

F

Face, angioma of, 129
Fallopian tubes, fibroids of, 206
Fibromata, ovarian, 205
Fibromyxoma, ovarian, 210
Fischer, Louis, Observations on infantile typhoid from the babies' ward of the Sydenham Hospital, 242
Flexner, Simon, Antimeningitis serum and the results of its employment, 1
Forearm, injury of, 143
Foreign bodies in œsophagus and bronchi, 272
Frambœsia tropica, 262
Fredet, Pierre, Treatment of cancer by fulguration, 22
Fulguration treatment of cancer, 22

G

Goitre, unilateral cystic, 131
Goodman, Henry M., The duty of the physician in the prevention of organic disease of the heart, 114
Gouty phlebitis, 108
Gravity method of removing adherent placenta, 199
Guisez, G., Diagnosis of foreign bodies in the œsophagus and bronchi, 272

H

Hamman, Louis, The use of tuberculin in treatment, 31
Heart disease, duty of the physician in the prevention of organic, 114
in infantile typhoid, 243
Hemorrhage from spleen after prolonged radiotherapy, 280
Hernia, radical cure of, followed by epiploitis, 145
Hymenolepis nana in the United States with report of two cases, 252
symptoms of, 258
Hypnotism and psychotherapy, 60
indications for, 62
Hysteria, hypnotism in, 63

I

Infantile typhoid, observations on, in the babies' ward, from the Sydenham Hospital, 242
Insanity, hypnotism in, 64
Insomnia, hypnotism in, 62
Intestines, chronic obstruction of, 141
Intra-omental cyst, 125

L

Larynx, death due to leukæmic infiltration of the, 280
Lee, W. Estell, The use of ethyl chloride as a general anæsthetic, 182
Leonard, Charles Lester, The Röntgen diagnosis of pulmonary tuberculosis, 189
Leukæmic infiltration of the larynx, death due to, 280
priapism, 286
Lip, squamous carcinoma of, 130
Liver in pernicious anæmia, 74
sarcoma of, 300
Longenecker, C. B., Color photography in relation to surgery, 166
Lungs, calcified tuberculosis of, 300
Röntgen diagnosis of tuberculosis of, 189
Lupus vulgaris, 175
Lymph-glands in pernicious anæmia, 74
Lymphosarcoma, retroperitoneal, 136

M

Male fern in Tænia nana, 253
Mapes, C. C., Ovarian fibromata, 205
Mediastinum, solid teratoma of, 296
Meningitis, chronic epidemic cerebrospinal, 11
epidemic cerebrospinal, 1
posterior basic, 13
tuberculous, 14
Mercury in adrenal disease, 97
in yaws, 271
Mitral regurgitation, 118
Morphinomania, hypnotism in, 63
Music in health and disease, 66

Myelæmia, 283
Myeloid thromboses in the corpora cavernosa, 280

N

Nerve suture, 143
Nervous system in adrenal insufficiency, 90
Niles, Geo. M., A general consideration of the therapeutic uses of diet, 54

O

Obesity, diet in, 56
Œsophagoscopy, 278
Œsophagus and bronchi, foreign bodies in, 272
Omentum, cyst of, 125
infection of, after hernia operations, 148
resection of, 163
Ovarian fibromata, 205

P

Papilloma of bladder, 142
Paraplegia in pernicious anæmia, 72
Parturition, complications of, 200
Perineal prostatectomy, 235
Perityphlitis, 134
Pernicious anæmia, blood in, 70
causes of, 68
pathology of, 69
the diagnosis and treatment of, 67
Phlebitis, gouty, 108
Pigmentation in Addison's disease, 92
Pisek, Godfrey Roger, Notes on the diagnosis and treatment of congenital syphilis, 247
Placenta, adherent, 199
Posterolateral spastic sclerosis in pernicious anæmia, 77
Priapism, persistent post mortem, 280
Prostatectomy, 233
Prostate, hypertrophy of, 229
indications for surgery of, 228
Prostatitis, chronic, 230
Psychotherapy, 60
Pulse, factor in prognosis of advanced tuberculosis, 103
Pylorus, carcinoma of, 138

R

Radiotherapy, fatal hemorrhage from spleen after prolonged, 280
Rectenwald, J. J., Gravity method of removing adherent placenta, 199
Regurgitation, aortic, 118
mitral, 118
Report of fifteen cases operated upon in Professor W. L. Rodman's Surgical Clinic at the Medico-Chirurgical Hospital, 125
Respiratory factor in prognosis of advanced tuberculosis, 103
Retroperitoneal lymphosarcoma, 136
Rheumatism and heart disease, 114
Rodman, J. Stewart, Report of fifteen cases operated upon in Professor W. L. Rodman's Surgical Clinic at the Medico-Chirurgical Hospital, 125
Röntgen diagnosis of pulmonary tuberculosis, 189
rays in congenital syphilis, 249
in the diagnosis of foreign bodies in œsophagus and bronchi, 274

S

Sarcoma of brain, 127
of liver, 300
of mediastinum, 300
Sclerosis, posterolateral spastic, in pernicious anæmia, 77
Sergent, Emile, Adrenal insufficiency, 88
Sex, factor in prognosis of advanced tuberculosis, 100
Shock, prevention of, 196
Silk ligatures, 146
Skin diseases, hypnotism in, 63
eruptions in congenital syphilis, 249
eruptions in yaws, 268
Spinal cord in pernicious anæmia, 76
Spleen, hemorrhage from, after prolonged radiotherapy, 280
in infantile typhoid, 243
in pernicious anæmia, 74
Stanton, William B., The prognosis in advanced tuberculosis, 99
Stenosis, aortic, 118
Stomach, ulcer of, 140
Suprapubic prostatectomy, 234
Surgery and color photography, 166
Syphilis and heart disease, 119
and yaws, 264
congenital, 247
treatment of congenital, 250

T

Tænia nana, 252
Temperature factor in prognosis of advanced tuberculosis, 102
Teratoma of mediastinum with history and autopsy notes, 296
Thromboses in the corpora cavernosa, myeloid, 280
Thyroid gland, cyst of, 131
Treponema pertenuis, 266
Tuberculin preparations, 35

Tuberculin reaction, 45
use of, in treatment, 31
Tuberculosis, diet in pulmonary, 58
of lungs, 300
prognosis in advanced, 99
Röntgen diagnosis of pulmonary, 189
treatment of incipient, 33
Tuberculous meningitis, 14
Typhoid, infantile, 242
symptoms of infantile, 243

U

Ulcer of stomach, 140
Urine factor in prognosis of advanced tuberculosis, 103
in infantile typhoid, 244
Uterus, early diagnosis of cancer of, 193

W

Wainwright, John W., Hypnotism and psychotherapy, 60
Warthin, Aldred Scott, Death due to leukæmic infiltration of the larynx; priapism persistent post mortem, myeloid thromboses in the corpora cavernosa; fatal hemorrhage from spleen after prolonged radiotherapy, 280
Widal reaction in infantile typhoid, 242
Woolley, Paul G., Frambœsia tropica, 262

Y

Yaws, 262
and syphilis, 264
treatment of, 271

GENERAL INDEX

(NINETEENTH SERIES)

Volumes are indicated by Roman numerals: March, i; *June*, ii; *September*, iii; *December*, iv.

A

Abdomen, surgery of, i, 288
Abscess, appendiceal, i, 95
in hip disease, ii, 177
in otitis media, extradural, iii, 245
of liver, i, 298
of lung, postoperative, iii, 129
Abscesses in typhoid fever, 34
Acetanilid poisoning in typhoid fever, iii, 36
Acetone in diabetic urine, tests for, ii, 104
Acetonuria, ii, 104
Acholuric jaundice, ii, 67, 80
Acroparæsthesia, iii, 51
Acute yellow atrophy in pregnancy, i, 128
of liver, i, 252
Addison's disease, iv, 88, 91
Adenofibroma of breast, iv, 132
Adenoma of liver, i, 298
Adherent placenta, gravity method of removal of, iv, 199
Adhesions, postoperative, iv, 141
Adhesive intestinal obstruction and pregnancy, i, 134
Adhesive plaster treatment in pleurisy, ii, 53
Adrenal disease and arteriosclerosis, i, 256
and kidney disease, i, 256
extract in adrenal disease, iv, 97
gland and mental development, iii, 255
function of, i, 240
insufficiency, iv, 88
acute, iv, 93
Adrenalin, i, 244
in pleural effusion, i, 244
in treatment of shock, iii, 118
Age, factor in prognosis of advanced tuberculosis, iv, 100
incident of typhoid fever patients, iii, 27
Aged, operations on the, i, 126
Air in chronic pelvic disease, fresh, iii, 181
insufflation in pleural effusion, i, 221
in surgery, hot, i, 289
Alcohol as a food, iv, 59
Alcoholic injections in neuralgia, i, 226
neuritis, i, 41
Alglare, P., Early and complete resection of varicose veins of the leg, iii, 154
Alimentary glycosuria, ii, 89
Alkalies in hyperchlorhydria, iii, 65
Allyn, Herman B., Tuberculous serofibrinous pleurisy and its treatment, ii, 44
Amaurosis in typhoid fever, iii, 30
Amœbic dysentery, i, 40, iii, 280
bismuth in, i, 225
ipecac in, i, 224
Amyl nitrite in hæmoptysis, i, 221
Anæmia and operations, i, 121
cholesterin in pernicious, i, 211, 258
diagnosis and treatment of pernicious, iv, 67
Anæsthetic, ethyl chloride as a general, iv, 182
Anaphylaxis, i, 255
Anatomy and surgery, i, 268
Anchylosis, congenital, iii, 193
Aneurism of cerebral vessels, ii, 280
Angina pectoris, treatment of, i, 218
Angioma of face, iv, 129
Anorectal fistula, ii, 238
treatment of, ii, 242
Anorexia nervosa, diet in, iv, 57
Anthrax, i, 230
Anti-anthrax serum, i, 231
Antidiphtheritic serum, sudden death after, i, 203
Antidysenteric serum, i, 224, iii, 280
Antimeningitis serum, i, 204
and the results of its employment, iv, 1
indications for the employment of, iv, 7
manner of administration of, iv, 8
manner of therapeutic action of, iv, 5
preparation and standardization of, iv, 2
results of, iv, 14
Antimony in acute bronchitis, i, 219
Antiperistaltic waves, i, 247
Antipneumococcic serum, i, 21
Antiscarlatinal serum, i, 201
Antisyphilitic treatment in tuberculosis, i, 208
Antitetanic serotherapy, iii, 11
serum, i, 229

Antithyroid serum in exophthalmic goitre, i, 214
Antityphoid inoculation i, 198, 233
Anus, imperforate, ii, 119
Aortic regurgitation, iv, 118
and operations, i, 117
stenosis, iv, 118
Apomorphine in bronchopneumonia, ii, 26
Appendicitis, acute, i, 289, iv, 134
and kidney lesions, i, 294
and pelvic lesions, i, 294
and tetany, i, 293
and trauma, i, 295
castor oil in, i, 289
causes of, i, 295
chronic, iii, 168, 173
in typhoid fever, iii, 36
leucocyte count in, i, 291
Rovsing's symptom in, i, 290
suppurative, i, 93
Appendicostomy, iv, 82
Appendix, carcinoma of, i, 296
diverticulum of, i, 295
gangrene of, iii, 164
Arrythmia, cardiac, i, 237
Arsenic in chorea, i, 226
in chronic pelvic disease, iii, 187
in exophthalmic goitre, i, 214
in pernicious anæmia, iv, 86
Arterial tension, diet in high, i 219
treatment in high, i, 219
Arteries pathology of, ii, 276
Arteriosclerosis and adrenal disease, i, 255
of cerebral vessels, ii, 279
Arthralgia, tubercular, i, 64
Arthritis deformans, etiology of, i, 102
excision of hip-joint in, i, 100
pathology, symptoms and treatment of, i, 101
tubercular, i, 67
Asman, Bernard, Anorectal fistula and its treatment, ii, 238
Atrophy of liver, acute yellow, i, 128, 252
Atropine in angina pectoris, i, 218
Autointoxication, diet in, iv, 58
Autoserotherapy in pleurisy, ii, 54

B

Bacillary dysentery, iii, 279
Barach, Joseph H., Clinical observations in five hundred cases of typhoid fever, iii, 24
Baths in chronic pelvic diseases, iii, 181
in scarlet fever, i, 201
Becker, E. W. and Carey, H W., A solid teratoma of the mediastinum with history and autopsy notes, iv, 296
Belladonna in hyperchlorhydria, ii, 65
Beta-oxybutyric acid, test for, ii 105
Bierring, Walter L., The diagnosis and treatment of pernicious anæmia, iv, 67
Bile-ducts, congenital obliteration of, iii, 72
Bilocular stomach, causes and symptoms of, iii, 138
pathology, diagnosis and surgical treatment of, iii, 140
Bismuth subnitrate in amœbic dysentery, i, 225
Bladder, papilloma of, iv, 142
prolapse of, iii, 177
Blood, coagulation of, i, 257
in diabetes, ii, 98
in infantile typhoid, iv, 242
in jaundice, ii, 67
in leukæmia, iv, 281
in pernicious anæmia, iv, 69
in typhoid fever, iii, 29
tubercle bacilli in, ii, 51
Bloodgood, Joseph C., Surgery, i, 266
Blood-pressure, i, 240, 271
and kidney diseases, i, 254
and kidney extract, i, 254
in typhoid fever, iii, 35
Blood-serum in hæmophilia, normal, i, 213
Blood-vessels of base of brain, ii, 293
pathology of, ii, 277
Bone-marrow cysts, i, 283
in pernicious anæmia, iv, 69
tumors, i, 282
Bone tumors, i, 281
Brain, abscess of, in suppurative otitis media, iii, 247
congestion of, and retarded mental development, iii, 258
embolism of, ii, 163
sarcoma of, iv, 127
Breast, adenofibroma of, iv, 132
carcinoma of, i, 263, iv, 133
carcinoma, cysts of, i, 285
cystic tumors of, i, 286
surgery of, i, 285
Bromides in epilepsy, i, 227
Bronchi and œsophagus, foreign bodies in, iv, 272
Bronchitis, antimony in acute, i, 219
in typhoid fever, iii, 30
nitric acid fumes in chronic, i, 220
postoperative, iii, 129
potassium iodide, oil of turpentine, balsam of Peru in chronic, i, 220
Bronchopneumonia in children, ii, 20
in gonococcic septicæmia, iii, 61
Bronchoscopy, iv, 278
Bubonic plague, i, 234
Buchanan, Leslie, Intra-ocular tumors, ii, 209
Buerger, Leo, The association of migrating thrombophlebitis with thromboangeitis obliterans, iii, 84
Bushnell, F. G. and Fletcher, H. N., Perithelioma testis, ii, 267

C

Cachexia and operations, i, 124
Cactus in Basedow's disease, iii, 51
Cæcum, perforation of, iv, 134
Cammidge reaction, present status of the, ii, 167
Calcium in tetany, i, 224, 240
Cancer and heredity, i, 259
and immunity, i, 262
and kraurosis vulvæ, ii, 195
chemistry of, i, 264
cysts of breast, i, 285
endemicity of, i, 259
experimental studies of, i, 274
parasitic origin of, i, 260
patients, blood serum of, i, 264
Carcinoma, i, 258
and gastroptosis, iii, 234
and operations, i, 124
of appendix, i, 296
of breast, iv, 133
of lip, squamous, iv, 130
of prostate, iv, 238
of pylorus, iv, 138
of uterus, early diagnosis of, iv, 193
of uvea, ii, 209
treatment of, by fulguration, iv, 22
Carey, H. W. and Becker, E. W., A solid teratoma of the mediastinum with history and autopsy notes, iv, 296
Carrière, Carron de la, Mineral waters in the treatment of syphilis, ii, 14
Caruncle of urinary meatus, iii, 172, 174
Castor oil in appendicitis, i, 290
in bronchopneumonia, ii, 26
Cataract, diabetic, ii, 12
Caulophyllum in chronic pelvic disease, iii, 188
Cellulose in diabetes mellitus, i, 209
Cerebellum, abscess of, iii, 236
Cerebral disorders, psychometric methods in diagnosis of, ii, 249
hemorrhage, pathogenesis of spontaneous, ii, 271
Cerebrospinal fever, antimeningitis in, i, 204
Chancroid of the vulva, ii, 207
Chemical controls of human body, i, 243
Children and operations, ii, 126
diagnosis and treatment of pneumonia in, ii, 20
Chloroform, death due to, ii, 128
injections in neuralgia, i, 227
Cholangitis, congenital, ii, 72
Cholecystitis, iv, 137
in typhoid fever, iii, 37
Cholelithiasis, iv, 137
Cholesterin in pernicious anæmia, i, 211 258
Chorea, arsenic in, i, 226
magnesium sulphate in, i, 226
treatment of, i, 225
Chorea, trional in, i, 225
Chorioid, leukosarcoma of, ii, 216
Ciliary body, tumors of, ii, 215
Cimicifuga in chronic pelvic disease, iii, 188
Circulation in adrenal insufficiency, iv, 89
Cirrhosis, biliary, ii, 77
Clothing in chronic pelvic disease, proper, iii, 181
Cod liver oil in rhachitis, i, 210
Cohen, Solomon Solis, Graves's disease; Raynaud's disease and some of the allied forms of vasomotor disorder (vasomotor ataxia), iii, 40
Colitis, ii, 143
Collapse after operations, iii, 114
Colon, congenital idiopathic dilatation of the, ii, 119
diagnosis and differential diagnosis of, ii, 133
etiology and pathology of, ii, 140
symptoms and prognosis of, ii, 134
treatment of, ii, 137
idiopathic dilatation of the, iii, 196
in gastroptosis, position of, iii, 220
Colonic irrigation in pernicious anæmia, iv, 85
Color photography, history of, iv, 67
in relation to surgery, iv, 166
technic of, iv, 171
Coma, diabetic, ii, 96
Compressed-air disease, i, 256
Congenital bilocular stomach, iii, 138
dilatation of colon, iii, 196
joint deficiencies (multiple), iii, 192
syphilis, iii, 271, iv, 247
Conjugal diabetes, ii, 88
Connective-tissue tumors, benign or malignant, i, 280
Constipation as cause of dilatation of colon, iii, 199
in chronic pelvic disease, iii, 184
in infants, ii, 133
Cornea, inflammation of, iii, 291
Corner, Edred M., Suppuration in appendicitis, i, 93
Corpora cavernosa, myeloid thromboses in, iv, 280
Coryza in infants, iv, 248
Cough in pleurisy, treatment of, ii, 53
Cullen, Thomas S., The early diagnosis of cancer of the uterus; operative technic, iv, 193
Cumston, Charles Greene, A study on the pathology and surgical tr atment of bilocular stomach, iii, 155; Epiploitis following the radical cure of inguinal hernia, iv, 145; Intestinal obstruction during pregnancy and the postpartum, i, 133

Cutaneous tuberculin tests, i, 236
Cyst, multilocular, iii, 172
Cystic tumors of breast, i, 286

D

Daniel, Peter, Congenital idiopathic dilatation of the colon (Hirschsprung's disease), ii, 119
Deaderick, William H., Hymenolepis nana in the United States with report of two cases, iv, 252
Deaver, John B., The indications for surgery of the prostate, iv, 228
Death rate and tuberculosis, i, 10
Dechaux and Gougèt, On cases of unforeseen death in scarlet fever, iii, 204
Deciduoma, i, 261
Delusions, hypnotism in, iv, 62
Dench, Edward Bradford, The intracranial complications of acute and chronic suppurative otitis media, iii, 240
Diabetes mellitus, ii, 86, 90
Cammidge reaction in, ii, 175
cellulose in, i, 209
diagnosis of, ii, 107
etiology of, ii, 87, 91
pancreatic extract in, i, 209
pathology of, ii, 92
potato starch in, i, 209
prognosis of, ii, 107
symptoms of, ii, 93
treatment of, ii, 108
Diabetic coma, ii, 96
treatment of, ii, 108
Diacetic acid, tests for, ii, 105
Diarrhœa in pernicious anæmia, iv, 84
in typhoid fever, iii, 31
Diazo reaction in typhoid fever, iii, 28
Diet, general considerations of the therapeutic uses of, iv, 54
in chronic pelvic disease, iii, 181
in diabetes mellitus, i, 209, ii, 109
in gout, i, 211
in high arterial tension, i, 219
in hyperchlorhydria, ii, 64
in rhachitis, i, 210
in scarlet fever, i, 201
in tuberculosis, iii, 6
Dieulafoy, G., Gonococcic septicæmia, iii, 59
Digestion factor in prognosis of advanced tuberculosis, iv, 103
in adrenal insufficiency, iv, 89
physiology of, i, 245
Digitalis in chronic valvular disease, i, 216
Dilatation of colon, congenital, ii, 119
Diller, Theodore, Chronic constitutional headaches, iii, 259
Dionin, iii, 274
Diphtheria, i, 202
pyocyanase in, i, 202
Dipsomania, hynotism in, iv, 63
Displacement of organs in pleurisy, ii, 48
Diuretics in pleurisy, ii, 54
Diverticulum of appendix, i, 295
Duckworth, Sir Dyce, Gouty phlebitis, iv, 108
Dumarest, F., Surgical pneumothorax as a treatment for phthisis, ii, 153
Dysentery, amœbic, iii, 280
bacillary, iii, 277
due to intestinal worms, iii, 288
exotic, iii, 277
Duodenitis, i, 252
Duval and Vinard, Sporotrichosis, i, 174
Dysentery, amœbic, i, 40, iii, 280
bacillary, iii, 277
due to intestinal worms, iii, 288
exotic, iii, 277
ipecac in amœbic, i, 224
polyvalent serum in, i, 224
Dysmenorrhœa, iii, 174
Dyspepsia, hypnotism in, iv, 63
Dyspnœa, in pleurisy, treatment of, ii, 53

E

Ear, suppurative diseases of middle, iii, 240
Earache and facial palsy, ii, 231
Earle, Samuel T., Report of a case of tubercular stricture of the rectum, with excision, i, 163
Eclampsia, pleural, ii, 164
Edsall, David T. and Nisbet, Verner, Medicine, i, 228
Eisendrath, Daniel N. and Straus, David C., Some postoperative complications, iii, 114
Electricity in chronic pelvic disease, iii, 188
Ellis, A. G., The pathogenesis of spontaneous cerebral hemorrhage, ii, 271
Embolism of lung, postoperative, iii, 129
Empyema, postoperative, iii, 129
Endocarditis in typhoid fever, iii, 35
mitral, iii, 60
rheumatic, iv, 115
Enteroptosis and Röntgen rays, iii, 217
Eosinophiles in Hymenolepis nana, iv, 258
Epididymitis, iv, 240
Epilepsy, bromides in, i, 227
Jacksonian, iv, 128
Epiploitis, diagnosis of, iv, 161
following the radical cure of hernia, iv, 145
pathology and symptoms of, iv, 150
treatment of, iv, 162
Epithelial tumors of skin and mucous membranes, i, 276
Erysipelas in typhoid fever, iii, 30

Erysipelas, magnesium sulphate in, i, 204
Eserine as laxative before operations, i, 288
Ethyl chloride as a general anæsthetic, iv, 182
Exercise in chronic pelvic disease, iii, 180
in diabetes, ii, 116
Exophthalmic goitre, iii, 40, 106
and the thymus gland, i, 242
and the thyroid gland, i, 242
antithyroid serum in, i, 215
arsenic in, i, 214
complications of, iii, 108
hypophysis extract in, i, 215
lecithin in, i, 215
operations in, i, 214, 216, iii, 108
quinine hydrobromide in, i, 215
Röntgen rays in, i, 214
sanitarium treatment in, i, 214
symptoms and diagnosis of, iii, 106
types of, i, 242
Extradural abscess, iii, 246
Eye, tumors of, ii, 209

F

Face, angioma of, iv, 129
nerve grafting in paralysis of, i, 86
Facial nerve, course of, ii, 226
paralysis, ii, 220
treatment of, ii, 224
Fæces in gastric dilatation, i, 75
tubercle bacilli in, ii, 51
Fallopian tubes, fibroids of, iv, 206
Fæcal fistula after appendicitis, i, 96
Fever in appendicitis, i, 95
in lobar pneumonia, ii, 33
Fibromata, ovarian, iv, 205
Fibromyxoma, ovarian, iv, 210
Fischer, Louis, Diagnosis and treatment of pneumonia in children, ii, 20; Observations on infantile typhoid from the babies' ward of the Sydenham Hospital, iv, 242
Fistula, anorectal, ii, 235
vesicovaginal, iii, 175
Fleas in Bubonic plague, i, 234
Fletcher, H. N. and Bushnell, F. G., Perithelioma testis, ii, 267
Flexner, Simon, Antimeningitis serum and the results of its employment, iv, 1
Flick, Lawrence F., The hospital for advanced cases of tuberculosis, i, 1
Forearm, injury of, iv, 143
Foreign bodies in œsophagus and bronchi, iv, 272
Fowler position, iii, 167
Frambœsia tropica, iv, 262
Francine, A. P., Treatment of tuberculosis, iii, 1
Fredet, Pierre, Treatment of cancer by fulguration, iv, 22
Freeman, Leonard, Nerve grafting in facial paralysis; report of a case, i, 86
Fulguration treatment of cancer, iv, 22

G

Gall-stones and Cammidge reaction, ii, 174
causation of, i, 250
formation of, i, 251
Gangrene of appendix, general peritonitis due to, iii, 164
Gastric dilatation, acute, iii, 127
Gastro-anastomosis, iii, 146
Gastro-enterostomy, iii, 147
Gastrohepatic ligament in gastroptosis, iii, 225
Gastro-intestinal radiologic examinations, iii, 217
Gastromesenteric ileus, iii, 125
Gastroplasty, iii, 143
Gastroptosis and Röntgen rays, iii, 217
treatment of, iii, 235
Gelatin in typhoidal hemorrhage, i, 199
Gigantism in Hirschsprung's disease, ii, 144
Glycosuria, ii, 88
alimentary, ii, 89
and operations, i, 120
nervous, ii, 89
renal, ii, 90
suprarenal, ii, 90
Goitre, exophthalmic, iii, 106
unilateral cystic, iv, 131
Gonococcic septicæmia, iii, 59
vaccine, iii, 62
Goodman, Henry M., The duty of the physician in the prevention of organic disease of the heart, iv, 114; The present status of the Cammidge reaction, ii, 167.
Gougèt and Dechaux, On cases of unforeseen death in scarlet fever, iii, 204
Gout, diet in, i, 211
Gouty phlebitis, iv, 108
Graves's disease, iii, 40
larval forms of, iii, 50
treatment of, iii, 57
Gravity method of removing adherent placenta, iv, 108
Grayson, Charles Prevost, Some reflections concerning the etiology and treatment of the neurovascular disturbances of the nose: hay fever, paroxysmal sneezing and recurrent rhinorrhœa, i, 167
Guisez, G., Diagnosis of foreign bodies in the œsophagus and bronchi, iv, 272
Gymnastics in pleurisy, pulmonary, ii, 55

H

Hæmatemesis, postoperative, iii, 119
Hæmolytic reaction in cancer, i, 264

Hæmophilia and operations, i, 123
normal blood-serum in, i, 213
Hæmoptysis, amyl nitrite in, i, 221
as indication to produce pneumothorax, ii, 165
Hallucinations, ii, 37
Hamman, Louis, The use of tuberculin in treatment, iv, 31
Hanot's disease, ii, 77
Hay fever, i, 167
Headache in Graves's disease, iii, 50
Headaches, chronic constitutional, iii, 259
Heart, arrythmia of, i, 237
block, i, 238
disease, duty of the physician in the prevention of organic, iv, 114
and operations, i, 116
treatment of chronic, i, 216
in cerebral hemorrhage, pathologic changes in, ii, 295
in infantile typhoid, iv, 243
in scarlet fever, paralysis of, iii, 205
in typhoid fever, iii, 35
nervous affections of, i, 239
Heat and disease, vi, 256
Hemorrhage and operations, i, 121
postoperative, iii, 119
treatment of, iii, 123
from spleen after prolonged radiotherapy, iv, 280
in typhoid fever, i, 199, iii, 31
pathogenesis of spontaneous cerebral, ii, 271
Hemorrhoids, iii, 164
Hepatectomy, i, 299
Hereditary jaundice, ii, 68
Heredity and cancer, i, 259
Hernia, inguinal and pregnancy, iii, 173
radical cure of, followed by epiploitis, iv, 145
umbilical, iii, 175
Hibbit, Charles W., The diagnosis of pelvic disease in women, ii, 200
Hip disease, abscess in, ii, 177
Hip-joint, in arthritis deformans, excision of, i, 100
Hirschsprung's disease, ii, 119, iii, 196
Hodgkin's disease, i, 283
Hormions, i, 243
Hospital for advanced cases of tuberculosis, i, 1
Howard, Campbell F., Mikulicz's disease and allied conditions, i, 30
Hydatid cyst of liver, i, 298
Hydrocephalus, internal, iii, 256
Hydrochloric acid in diseases of stomach, i, 248
Hydrotherapy for chronic rheumatism, i, 38
Hymenolepis nana in United States with report of two cases, iv, 252
symptoms of, iv, 258
Hyperchlorhydria, ii, 59
Hyperchlorhydria and Cammidge reaction, ii, 174
causes and symptoms of, ii, 59
diagnosis and prognosis of, ii, 63
treatment of, ii, 63
Hypnotism, ii, 38
and psychotherapy, iv, 60
indications for, iv, 62
Hypoglossal anastomosis with facial nerve, i, 90
Hypophysis extract in exophthalmic goitre, i, 215
Hysteria, hypnotism in, iv, 63
Hysterical pseudo-appendicitis, i, 292

I

Icterus neonatorum, ii, 71
Ileus, postoperative, iii, 128
Immunity and cancer, i, 262
in tuberculosis, i, 262
Immunization against typhoid fever, ii, 1
Infantile typhoid, observations on, from the babies' ward of the Sydenham Hospital, iv, 242
Infarct of lung, postoperative, iii, 129
Influenza, bacteriology of, i, 228
Influenzal meningitis, i, 228
Inguinal hernia, iii, 173
epiploitis following the radical cure of, iv, 145
Inhalation treatment of bronchopneumonia, ii, 26
Inoscopy, ii, 51
Insanity, after typhoid fever, iii, 36
hypnotism in, iv, 64
Insomnia, hypnotism in, iv, 62
Intestinal worms, iii, 288
Intestines, chronic obstruction of, iv, 141
obstruction of, i, 301
during pregnancy and the postpartum, i, 133
Intracranial complications of otitis media, iii, 240
Intra-ocular tumors, ii, 209
Intra-omental cyst, iv, 125
Intussusception and pregnancy, i, 137
Iodine contents of thyroid gland, i, 241
Ipecac in amœbic dysentery, i, 224
Iris, tumors of, ii, 215
Iron in chronic pelvic disease, iii, 186
Isolation for tuberculosis, i, 5
Itching in diabetes, treatment of, ii, 117

J

Jardine, Robert, A case of acute yellow atrophy of the liver and pernicious vomiting of pregnancy, i, 128
Jaundice, acute catarrhal, i, 251
with splenomegaly, ii, 67
Joint deficiencies, congenital multiple, iii, 192

K

Kelsall, Oliver H., Rupture of urethra, acute urinary retention, secondary syphilis, i, 155
Keratitis, interstitial, iii, 271
Kidney complications, postoperative, ii 132
disease and adrenal disease, i, 255
and operations, i, 118
extract and blood pressure, i, 254
lesions and appendicitis, i, 294
Kidneys, causes of chronic inflammation of, i, 253
diseases of, during scarlet fever, iii, 211
in typhoid fever, iii, 35
extract of, in nephritis, i, 254
in cerebral hemorrhage, pathologic changes in, ii, 296
influence of inflammation of, on gastric secretion, i, 250
internal secretion of, i, 253
results of removal of, i, 254
treatment of inflammation of, i, 222
Kraurosis vulvæ, ii, 190
treatment of, ii, 196

L

Lachrymal glands, enlargement of, i, 30
Lagane, L., The present position of antitetanic serotherapy, iii, 11
Landouzy, L., Typhobacillosis, i, 79
Larynx, death due to leukæmic infiltration of the, iv, 280
sporotrichosis of, i, 177
Lavage in acute gastric dilatation, i, 250
in hyperchlorhydria, ii, 65
Laxatives in chronic pelvic disease, iii, 184
Lecithin in exophthalmic goitre, i, 215
Lederman, Isaac, Interstitial keratitis, iii, 271
Lee, W. Estell, The use of ethyl chloride as a general anæsthetic, iv, 182
Leonard, Charles Lester, The Röntgen diagnosis of pulmonary tuberculosis, iv, 189
Leptomeningitis following suppurative otitis media, iii, 251
Lericke and Poncet, A., Acute tubercular rheumatism, i, 64
Leucocyte count in appendicitis, i, 291
in typhoid fever, iii, 29
Leucocytes in pleurisy, ii, 50
Leucocytic extract in pneumonia, i, 200
Leucoplakia of the vulva, ii, 192
Leucosarcoma of the chorioid, ii, 216
of the uvea, ii, 210
Leukæmia, i, 49
Röntgen rays in, i, 212
streptococcus and prodigiosus toxin in, i, 213
Leukæmic infiltration of the larynx, death due to, iv, 280
priapism, iv, 286
Lip, squamous carcinoma of, iv, 150
Liver, abscess of, i, 298
acute yellow atrophy of, i, 252
in pregnancy, i, 128
and Cammidge reaction, cirrhosis of, ii, 174
cirrhosis of, i, 41, 297
in gastroptosis, iii, 227
injuries and hemorrhage of, i, 297
in pernicious anæmia, iv, 74
sarcoma of, iv, 300
surgery of, i, 297
tumors of, i, 298
Longenecker, C. B., Color photography in relation to surgery, iv, 166
Loveland, Bradford C., Psychasthenia, ii, 36
Lung complications, postoperative, iii, 129
Lungs, calcified tuberculosis of, iv, 300
Röntgen diagnosis of tuberculosis of, iv, 189
Lupus vulgaris, iv, 175
Lymphadenoid hyperplasia, i, 53
Lymphatics and absorption, i, 184
Lymph-glands in pernicious anæmia, iv, 74
Lymphosarcoma, i, 262, 284
retroperitoneal, iv, 136

M

MacCallum, W. G., Absorption from the peritoneal cavity, i, 182
Magnesium sulphate in chorea, i, 226
in erysipelas, i, 202
in tetanus, i, 208
Male fern in Tænia nana, iv, 253
Mania in typhoid fever, iii, 36
Mapes, C. C., Ovarian fibromata, iv, 205
Massage in chronic pelvic disease, iii, 188
Mayo's method of curing umbilical hernia, iii, 175
of removing appendix, iii, 166
McCarthy, D. J., Neuropathology in childhood with a consideration of pathological factors in some cases of retarded mental development, iii, 253
Mediastinum, solid teratoma of, iv, 296
Melanoma of the uvea, ii, 210
Melanotic neoplasms, i, 280
Meningitis, chronic epidemic cerebrospinal, iv, 11
epidemic cerebrospinal, iv, 1
influenzal, i, 228
in suppurative otitis media, localized, iii, 245
posterior basic, iv, 13
tubercular, i, 82, iv, 14
Mental development, retardation of, iii, 253

Menorrhagia due to retroversion, iii, 174
Mercury and acute yellow atrophy, i, 252
in adrenal disease, iv, 97
in syphilis, iii, 23
in yaws, iv, 271
succinimide in tuberculosis, i, 208
Mesentery in Hirschsprung's disease, abnormally long, ii, 141
Mesmer and Perkins's tractors, iii, 16
Mikulicz's disease and allied conditions, i, 30
syndrome, i, 52
etiology and pathology of, i, 53
pathogenesis and treatment of, i, 56
Milk in typhoid fever, i, 232
Mineral waters in the treatment of syphilis, ii, 14
Miscarriage, high temperature following, iii, 171
Mitral regurgitation, iv, 118
Moles, benign and malignant pigmented, i, 279
Morphine in chronic heart disease, i, 218
Morphinomania, hypnotism in, iv, 63
Mortality in typhoid fever, iii, 39
Mucous membranes, epithelial tumors of, i, 276
sporotrichosis of, i, 177
Multilocular cyst, iii, 172
Muschlitz, Chas. H., Congenital joint deficiencies (multiple), iii, 192
Muscle, sporotrichosis of, i, 177
Music in health and disease, iv, 66
Myasthenia gravis and parathyroid glands, i, 249
Myelæmia, iv, 283
Myeloid thromboses in the corpora cavernosa, iv, 280
Myeloma, iv, 282
Myocarditis and operations, i, 118

N

Nails in exophthalmic goitre, iii, 49
Nativity in typhoid fever patients, iii, 25
Nephritis, postoperative, iii, 133
scarlatinous, iii, 211
Nerve grafting in facial paralysis, i, 86
suture, iv, 143
Nervous glycosuria, ii, 89
system in adrenal insufficiency, iv, 90
Neuralgia, alcohol injections in, i, 226
chloroform injections in, i, 227
Neurasthenia, ii, 36
Neuritis, alcoholic, i, 41
and diabetes, multiple, ii, 94
in typhoid fever, iii, 36
Neuropathic dilatation and hypertrophy of the colon, ii, 144
Neuropathology in childhood, iii, 253
Neurovascular disturbances of nose, i, 167
Niles, Geo. M., A general consideration of the therapeutic uses of diet, iv, 54; Some remarks on hyperchlorhydria, ii, 69
Nisbet, Verner, and Edsall, David L., Medicine, i, 228
Nitric acid fumes in chronic bronchitis, i 220
Nitrogen insufflation in pulmonary tuberculosis, ii, 155
Nose, neurovascular disturbances of, i, 167
Notes on cases at the Samaritan Hospital for Women, Montreal, iii, 164

O

Obesity, diet in, iv, 56
and operations, i, 126
Obstruction of intestines during pregnancy and the postpartum, i, 133
Occupation neuritis, i, 23
Occupations and so-called rheumatic pains, i, 16
Ochsner, Albert J., Exophthalmic goitre from the standpoint of the clinical surgeon, iii, 106
Ocular tuberculin test, i, 236
typhoidal reaction, i, 233
Œsophagoscopy, iv, 278
Œsophagus and bronchi, foreign bodies in, iv, 272
Oils, effect of, on gastric secretion, i, 249
Olive oil in gastric hyperacidity, i, 223
Omentum, cyst of, iv, 125
infection of, after hernia operations, iv, 148
resection of, iv, 163
Open-air treatment in pneumonia, i, 199
Operations, preparation for, i, 272
Operative treatment of exophthalmic goitre, i, 214
Opium in diabtes, ii, 117
Orange juice in rhachitis, i, 210
Otitis and facial palsy, ii, 223
media, intracranial complications of acute and chronic suppurative, iii, 240
Ovarian fibromata, iv, 205
region, pain in, ii, 200
Ovaries and deciduomata, i, 261
old maid's, iii, 173

P

Pain in pleurisy, treatment of, ii, 52
Palmer, Chauncey D., The constitutional treatment of chronic pelvic diseases, iii, 178
Pancoast, Henry K., The radiographic examination of the gastro-intestinal tract from a practical standpoint, especially in connection with the diagnosis and treatment of gastro-enteroptosis, iii, 217

Pancreas and diabetes, ii, 91
and hydrochloric acid i, 248
physiology of, i, 245
stimulating effect of dilute acids on, i, 243
surgery of, i, 300
tumors of, i, 301
Pancreatitic extract in diabetes mellitus, i, 209
insufficiency, i, 249
reaction, ii, 167
secretion in stomach, i, 248
test, i, 249
Pancreatitis, i, 300
hemorrhagic, ii, 172
Paralysis of face, nerve grafting in, i, 86
of the insane, ii, 264
refrigeratory facial, ii, 220
Paraplegia in pernicious anæmia, iv, 72
Parasitic origin of cancer, i, 260
Parasyphilis, ii, 259
Parathyroid gland, function of, i, 240
Paravertebral dulness in pleurisy, ii, 48
Parotid gland, swelling of, i, 38
Paroxysmal sneezing, i, 167
Parturition, complications of, iv, 200
Pellagra, i, 229
Pelvic disease of women, diagnosis of, ii, 200
diseases, constitutional treatment of chronic, iii, 178
lesions and appendicitis, i, 294
Pepsin in diseases of the stomach, i, 247
Perforation in typhoid fever, iii, 34
Pericarditis in typhoid fever, iii, 35
Perineal prostatectomy, iv, 235
Peristalsis, i, 246
Perithelioma testis, ii, 267
Peritoneal cavity, absorption from, i, 182
Peritonitis due to gangrene of the appendix, iii, 164
Perityphlitis, iv, 134
Perkins's tractors, iii, 16
Pernicious anæmia, blood in, iv, 70
causes of, iv, 68
cholesterin in, i, 211, 258
pathology, diagnosis and treatment of, iv, 67
vomiting of pregnancy, i, 128
Peru, balsam of, in chronic bronchitis, i, 220
Phenacetin poisoning in typhoid fever, iii, 36
Phlebitis, gouty, iv, 108
in typhoid fever, iii, 36
migrating, iii, 89
Phosphorus in rhachitis, i, 210
Physiology and surgery, i, 270
Pigmentation in Addison's disease, iv, 92
Pisek, Godfrey Roger, Notes on the diagnosis and treatment of congenital syphilis, iv, 247
Pituitary body, i, 244
Placenta, adherent, iv, 199
Pleural eclampsia, ii, 164
effusion, adrenalin in, i, 222
insufflation of air in, i, 221
Pleurisy, etiology and diagnosis of, ii, 46
gonococcic, iii, 67
in typhoid fever, iii, 31
postoperative, iii, 129
symptoms and treatment of, ii, 44
tuberculous serofibrinous, ii, 44
Pneumonia in children, diagnosis and treatment of, ii, 20
in typhoid fever, iii, 31
leucocytic extract in, i, 200
lobar, ii, 27
open air treatment of, ii, 199
postoperative, iii, 129
venesection in, i, 200
Pneumothorax as treatment for phthisis, surgical, ii, 153
Poncet, A. and Lericke, Acute tubercular rheumatism, i, 64
Posterolateral spastic sclerosis, in pernicious anæmia, iv, 77
Postoperative complications, iii, 114
Postpartum, intestinal obstruction during, i, 133
Potassium iodide in chronic bronchitis, i, 220
Potter, Philip S., Hirschsprung's disease, iii, 196
Pregnancy and inguinal hernia, iii, 173
intestinal obstruction during, i, 133
in typhoid fever, iii, 36
and operations, iii, 125
pernicious vomiting of, i, 128
tubal, iii, 169
Priapism, persistent post mortem, iv, 280
Proctoclysis in shock, saline, iii, 118
Prodigiosus and streptococcus toxin in leukæmia, i, 213
Prostate, hypertrophy of, iv, 229
indications for surgery of, iv, 228
Prostatectomy, iv, 233
Prostatitis, chronic, iv, 230
Pruritis vulvæ, ii, 192
Pseudo-angina in Graves's disease, iii, 52
Pseudo-appendicitis hysterica, i, 292
Pseudoleukæmia, i, 43
Pseudorheumatism, i, 16
Pseudotabes and diabetes, ii, 94
Psychasthenia, ii, 36
Psychology and surgery, i, 267
Psychometric methods in diagnosis of cerebral disorders, ii, 249
Psychotherapy, iv, 60
in chronic pelvic disease, iii, 190
Pulmonary abscess, postoperative, iii, 129
embolism, postoperative, iii, 129
infarct, postoperative, iii, 129
œdema in scarlet fever, iii, 211
Pulsatilla in chronic pelvic disease, iii, 187

Pulse, factor in prognosis of advanced tuberculosis, iv, 103
Pyloric spasm and gastroptosis, iii, 237
Pylorus, carcinoma of, iv, 138
position of, in gastroptosis, iii, 223
sphincter of, i, 246
Pyocyanase in diphtheria, i, 202
Pyuria and operations, i, 119

Q

Quinine in chronic pelvic disease, iii, 186
hydrobromide in exophthalmic goitre, i, 215

R

Radiographic examination of gastro-intestinal tract, iii, 217
Radiotherapy, fatal hemorrhage from spleen after prolonged, iv, 280
Raynaud's disease, iii, 40
Rectenwald, J. J., Gravity method of removing adherent placenta, iv, 199
Rectum, development of, ii, 150
dilatation of, ii, 127
tubercular stricture of, i, 163
Refrigeratory facial paralysis, ii, 220
Regurgitation, aortic, iv, 118
mitral, iv, 118
Reik, H. O., Refrigeratory facial paralysis: How is it produced? ii, 220
Relapses in typhoid fever, iii, 37
Renal glycosuria, ii, 90
Report of fifteen cases operated upon in Professor W. L. Rodman's Surgical Clinic at the Medico-Chirurgical Hospital, iv, 125
Respiration, physiology of, i, 244
Respiratory factor in prognosis of advanced tuberculosis, iv, 103
Rest in chronic pelvic diseases, iii, 179
cure in chronic pelvic diseases, Mitchell's, iii, 189
in tuberculosis, iii, 4
Retention, acute urinary, i, 155
Retinal elevation in uveal tumors, ii, 217
Retroperitoneal lymphosarcoma, iv, 136
Rhachitis, cod-liver oil in, i, 210
diet in, i, 210
orange juice in, i, 210
phosphorus in, i, 210
Rheumatic pains and occupations, i, 16
Rheumatism, acute tubercular, i, 64
and heart disease, iv, 14
subacute tubercular, i, 65
treatment of chronic, with nux vomica, i, 26
Rhinorrhœa, recurrent, i, 167
Richardson, F. A., Excision of the hip-joint in arthritis deformans, i 100
Rodman, J. Stewart, Report of fifteen cases operated upon in Professor W. L. Rodman's Surgical Clinic at the Medico-Chirurgical Hospital, iv, 125
Röntgen diagnosis of pulmonary tuberculosis, iv, 189
picture in gastric dilatation, i, 77
rays, examination of gastro-intestinal tract with, iii, 217
in congenital idiopathic dilatation of the colon, ii, 135
in congenital syphilis, iv, 249
in diagnosis of foreign bodies in œsophagus and bronchi, iv, 274
in exophthalmic goitre, i, 214
in leukæmia, i, 212
in pulmonary tuberculosis, ii, 160
Rovsing's symptom in appendicitis, i, 290
Rupture of urethra, i, 155

S

Salicylates in pleurisy, ii, 54
Salivary glands, enlargement of, i, 30
Sanitarium treatment in exophthalmic goitre, i, 214
of tuberculosis, i, 206
Saphenous vein, ligation of, iii, 155
Sarcoma, i, 280
of brain, iv, 127
of liver, iv, 300
of mediastinum, iv, 300
of the uvea, ii, 210
Scarlet fever, baths and diet in, i, 201
serum in, i, 201
unforeseen death in, iii, 204
Schwatt, H., The treatment of abscess in hip disease with a report of cases, ii, 177
Sclerosis, posterolateral spastic, in pernicious anæmia, iv, 77
Secretin, i, 243
Sensitization, i, 235
Septicæmia, gonococcic, iii, 59
typhoidal, i, 89
Sergent, Emile, Adrenal insufficiency, iv, 88
Sex, factor in prognosis of advanced tuberculosis, iv, 100
of five hundred typhoid fever patients, iii, 25
Shock, i, 270
after operations, iii, 114
and operations, i, 121
prevention of, iv, 196
treatment of, iii, 116
Shoemaker, Harlan, Immunization against typhoid fever, with a study of preventive inoculations, ii, 1
Silk ligatures, iv, 146
Sinus thrombosis in middle ear disease, iii, 241
Skin, anthrax of, i, 231
cancer of, i, 264
diseases, hypnotism in, iv, 63
epithelial tumors of, i, 276

Skin eruptions in congenital syphilis, iv, 249
in yaws, iv, 268
necrosis, after typhoid fever, iii, 36
sporotrichosis of, i, 175
Sneezing, paroxysmal, i, 167
Smith, A. Lapthorn, Notes of cases at the Samaritan Hospital for Women, Montreal, iii, 164
Sommerville, David, Diagnosis of gastric dilatation, i, 74
Spinal cord in pernicious anæmia, iv, 76
Spleen, hemorrhage from, after prolonged radiotherapy, iv, 280
in infantile typhoid, iv, 243
in pernicious anæmia, iv, 74
in typhoid fever, iii, 30
Splenomegaly, congenital familial, ii, 67, 80
Sporotrichosis, i, 174
Stanton, William B., The prognosis in advanced tuberculosis, iv, 99
Status lymphaticus, ii, 128
Stenosis, aortic, iv, 118
Stevens, A. A., Treatment, i, 198
Stokes-Adams disease, i, 238
Stomach, acute dilatation of, i, 250
after operations, iii, 125
and Cammidge reaction, carcinoma of, ii, 174
dilatation of, i, 74, iii, 230
evacuation of, i, 245
hour-glass contraction of, iii, 235
hyperacidity of, i, 223
influence of nephritis on secretion of, i, 250
of oil on secretion of, i, 249
lavage in acute dilatation of, i, 250
normal position of, iii, 219
operations on, iii, 143
pathology and treatment of bilocular, iii, 135
physiology of, i, 245
position of, in gastroptosis, iii, 222
symptoms of atonic dilatation of, i, 74
ulcer of, i, 223, iii, 136, iv, 140
Straus, David C. and Eisendrath, Daniel N., Some postoperative complications, iii, 114
Streptococcus and prodigiosus toxins in leukæmia, i, 213
Stricture of rectum, tubercular, i, 163
Strophanthin in chronic valvular disease, i, 217
Sulphur waters and syphilis, ii, 14
Suprapubic prostatectomy, iv, 234
Suprarenal gland in scarlet fever, iii, 213
Surgery and anatomy, i, 268
and color photography, iv, 166
and physiology, i, 270
and psychology, i, 267
Syphilis and heart disease, iv, 119
Syphilis and operations, i, 127
and yaws, iv, 264
congenital, iii, 271, iv, 247
secondary, i, 155
treatment of congenital, iv, 250

T

Taboparalysis, ii, 253
Tachycardia, iii, 48
paroxysmal, i, 239
Tænia nana, iv, 252
Temperature factor in prognosis of advanced tuberculosis, iv, 102
Teratoma of mediastinum with history and autopsy notes, iv, 296
Test meal, value of, i, 248
Testicle, perithelioma of, ii, 267
Tetanus antitoxin, i, 229
and its bacterium, i, 228
magnesium sulphate in, i, 208
treatment of, iii, 11
Tetany and appendicitis, i, 293
and parathyroid glands, i, 241
calcium salts in, i, 226
in typhoid fever, iii, 36
Thoracentesis in pleurisy, ii, 57
Thrombo-angeitis obliterans, iii, 84
Thrombophlebitis, association of, with thrombo-angeitis obliterans, iii, 84
without symptoms, iii, 86
with symptoms of limited vein involvement, iii, 87
Thromboses in the corpora cavernosa, myeloid, iv, 280
Thrombosis of lateral sinus, iii, 241
Thymus gland in scarlet fever, iii, 214
Thyroid gland, cyst of, iv, 131
enlargement of, iii, 48
and exophthalmic goitre, i, 242
function of, i, 240, 241
Thyroidectomy in exophthalmic goitre, i, 216
Tic, convulsive, operative treatment, i, 87
Torsion and pregnancy, i, 133
Transfusion in shock, saline, iii, 118
Treponema pertenuis, iv, 256
Trichocephalus dispar in bubonic plague, i, 234
Trional in chorea, i, 225
Tubal pregnancy, iii, 169
Tubercle bacilli in fæces and blood, ii, 51
bacillus in typhoid fever, iii, 37
Tubercular meningitis, i, 82, iv, 14
rheumatism, acute, i, 64
stricture of rectum, i, 63
Tuberculin, i, 206, 236, iii, 9
in bronchopneumonia, ii, 25
preparations, iv, 35
reaction, iv, 45
test, ii, 51
use of, in treatment, iv, 31
Tuberculosis and anorectal fistula, ii, 239
and operations, i, 127

Tuberculosis, antisyphilitic treatment in, i, 206
diet in pulmonary, iv, 58
hospital for advanced cases of, i, 1
immunity against, i, 236
mercury succinimide in, i, 208
ocular and cutaneous tests in, i, 236
of hip-joint, ii, 178
of lungs, iv, 300
prognosis in advanced, iv, 99
Röntgen diagnosis of pulmonary, iv, 189
sanitarium treatment of, i, 206
surgical pneumothorax as treatment of pulmonary, ii, 153
susceptibility to, i, 2
treatment of, i, 206, iii, 1
incipient, iv, 33
Tuberculous abscess, evacuation of, ii, 181
serofibrinous pleurisy, ii, 44, 49
Tumors, intra-ocular, ii, 209
necessity of removal of, i, 275
of liver, i, 298
Turpentine in chronic bronchitis, i, 220
Tympanotomy and facial palsy, ii, 235
Typhobacilliosis, i, 79
Typhoid-bacillus carriers, i, 232
Typhoid fever, i, 198, 231
clinical observations in 500 cases of, iii, 24
complications in, iii, 30
etiology of, iii, 25
hemorrhage in, i, 199
immunization against, ii, 1
infantile, iv, 242
metabolism in, i, 233
ophthalmodiagnostic tests in, i, 233
previous attacks in, iii, 28
symptoms of infantile, iv, 243
treatment of, iii, 37
vaccination against, i, 198

U

Ulcer of stomach, iv, 140
Umbilical hernia, iii, 175
Uræmial postoperative, iii, 133
Urethra, rupture of, i, 155
Urinary meatus, painful caruncle of, iii, 172, 174
retention, acute, i, 155
Urine factor in prognosis of advanced tuberculosis, iv, 103
in diabetes, ii, 99
in infantile typhoid, iv, 244
tests for sugar in, ii, 101
Urticaria, factitious, iii, 43
Uterus, early diagnosis of cancer of, iv, 193
prolapse of, iii, 177
retroversion of, iii, 173
menorrhagia due to, iii, 174
Uvea, diagnosis of tumors of, ii, 211
tumors of, ii, 209

V

Vaccination against bubonic plague, i, 234
against typhoid fever, i, 198, ii, 7
Vaccine, antigonococcic, iii, 62
preparation of, ii, 8
Vaginismus, iii, 174
Valvular dis., digitalis in chronic, i, 216
morphine in chronic, i, 218
strophanthin in chronic, i, 217
Varicose veins of leg, complete resection of, iii, 154
pathology of, iii, 159
Vasomotor disorders, iii, 40
Veins of leg, varicose, iii, 154
Venesection in pneumonia, i, 200
Vesicovaginal fistula, iii, 175
Vinard and Duval, Sporotrichosis, i, 174
Volvulus and pregnancy, i, 137
Vomiting of pregnancy, pernicious, i, 128
postoperative, iii, 123
Vomitus of gastric dilatation, i, 75
Vulva, chancroid of, ii, 207
kraurosis of, ii, 190

W

Wainwright, John W., Hypnotism and psychotherapy, iv, 60
Walsh, James J., Occupations and so-called rheumatic pains, i, 16; Women in Medicine, iii, 71
Warthin, Aldred Scott, Death due to leukæmic infiltration of the larynx; priapism persistent post mortem, myeloid thromboses in the corpora cavernosa; fatal hemorrhage from spleen after prolonged radiotherapy, iv, 280
Water in diabetes, ii, 115
Waterson, Davina, Mesmer and Perkins's tractors, iii, 16
Weber, F. Parkes, Congenital familial splenomegaly with chronic acholuric jaundice, ii, 67
Wells, Edward F., Diabetes, ii, 86
Widal react. in infantile typhoid, iv, 242
in typhoid fever, iii, 28
Williams, Tom A., Illustrations of the advantage of psychometric methods in diagnosis, prognosis and treatment of cerebral disorders, ii, 249
Willmoth, A. David, Conditions modifying operative work, i, 116
Women in Medicine, iii, 71
Woods, Richard F., Kraurosis vulvæ, with a report of two cases, ii, 190
Woolley, P. G., Exotic dysenteries, iii, 277
Worms, intestinal, iii, 288

Y

Yaws, iv, 262
and syphilis, iv, 264
treatment of, iv, 271

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